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# Lecture Notes

— ON —

# GENERAL PATHOLOGY,

BALTIMORE MEDICAL COLLEGE.

Session 1895-1896.

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# Errata.

Page 5. Line 7 from top read, "waste" for wase.

" 10. " 3 " bottom, read, "that is, it is" for that it is.

" 17. " 2 " " read, "it is not necessary" for it is necessary.

" 18 " 13. " bottom read, "and must" for amust.

" 24 " 6 " " read, "take" for takes

" 44 Top line read, "persists" for pre-exists.

" 56 Line 16 from top, read "the spindle shaped body" for Spindle shaped body.

" 61 Lines 12 and 13 from top read, "Osteomata" for osteoma.

" 62 Line 7 from top read, "the corpus cavernosum" for corpus cavernosum.

" 62 Last line, last word, read "those" for these

" 63. Line 11 from top, read "Sarcomata" for Sarcomas.

" 63. " 8 " bottom read, "Angeio-Sarcoma" for Angeioma-Sarcoma.

# Introductory Note

In preparing these brief notes of the lectures on pathology to be delivered to the Class during the session of 1895-1896, the lecturer wishes to clearly define their scope.

In the following pages, the lectures are by no means presented in full. This should be clearly understood; only the outline of each is given, and fly-leaves are left upon which it is hoped each student will note such facts gathered from the lectures and not stated in the text as appear to him to be of importance. If this is properly done the double purpose will be subserved, of guiding the hearer at the time of the lecture, and of enabling him to satisfactorily review from time to time the ground covered.

Each student is advised to take rough notes at the time of the lecture and afterward to place upon the fly-leaves of these notes such facts as appear to him to fill out the subject under discussion. In this way a fairly accurate knowledge of the various subjects presented should be obtained.

Delano Ames.

Pathological Laboratory,
Baltimore Medical College,
August 1st, 1895.

# INTRODUCTION.

DEFINITION: Pathology may be defined as the science of disease; that science which attempts to discover the causes of disease, their mode of action and the nature of the changes which they produce.

DIVISION OF THE SUBJECT:- From the above definition we may divide our subject into

1. Special Pathology.
2. General Pathology.

Special pathology is that portion devoted to the study of the various changes which have been produced in the gross and microscopic structure of different organs and tissues of the body by disease. It is thus analogous to anatomy and histology, which study the normal structure, gross and microscopic, of the organs and tissues.

General pathology on the other hand is that branch of the subject which like physiology, (which strives to discover the laws which govern the body in health) tries to find out the laws which govern the body in disease. Cohnheim called this branch of pathology "morbid physiology" General pathology is subdivided into a study of

1. The causes of disease, or etiology
2. The processes of disease.

We will devote most of our time to the second division.

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DEFINITION OF HEALTH: We may define health "as the maintenance of the living body in such a condition that neither the performance of its ordinary functions, or any slight disturbance of them interferes with its activity or leaves it worse than before". Of course the physiological functions may vary within wide limits, the perfect well being of the individual maintained, consequently our standard of health is not a rigid one.

DEFINITION OF DISEASE: The normal condition of the body is that of health, deviations from this normal constitute disease. We may therefore define disease "as a deviation from the normal vital processes" or "as a phase of life in which one or more of the physiological processes is running an abnormal course."

Diseases may be classified from the standpoint of their etiology into:

1. Those due to injury; using the word injury in its broadest sense, and by it meaning harm produced by any foreign agent whether it be the bite of a snake, the wound from a blow, the poison of some specific disease, or the results of exposure to cold, injudicious diet and the like.
2. Those due to congenital defects of structure.
3. Those due to the effects of wear and tear.
4. Those due to senile changes.

TISSUES AND CELLS: The body is composed of various tissues which in turn are made up of separate independent structures the cells, and of their derivatives, the intercellular substance, salts, etc.

CELLS: Are minute (microscopic) slightly granular, semi-translucent structures composed of:

1. A cell membrane.
2. Cell substance, or protoplasm, the living matter.
3. One or more nuclei.

The nuclei in their turn are composed of:

1. A nuclear net work.
2. Nuclear juice.
3. Nucleoli or small granular like bodies lying in the meshes of the nuclear net work.

The essential part of a cell is its protoplasm, which is a very complex substance composed of carbon, hydrogen, oxygen,

sulphur and sometimes phosphorus. Its structural formula is not known. The properties which it manifests and which taken together distinguish it absolutely from all forms of lifeless matter are:

1. Its chemical composition. (It can not be made in the laboratory)
2. Its powers of waste and repair, and of growth.
3. Its power of reproduction

With this brief introduction we will begin our study of pathology with a consideration of the disturbances of the circulation, and first take up Local Anaemia.

## LOCAL ANAEMIA.

DEFINITION: A condition resulting whenever the blood supply to an organ, or part is cut off, in part or entirely, by obstruction of the artery of supply, from whatever cause.

The condition is sometimes called ischaemia when the obstruction to the blood supply is only partial.

CAUSES: This condition may result from:

1. Undue increase of the natural arterial resistance.
2. Addition of new abnormal resistance.

Causes coming under the first head are for example:

a. Exposure to cold, causing a contraction of the arteries.

b. Irritation of the vaso-constrictor nerves from any cause resulting in a narrowing of the arteries supplied by the nerves irritated. This condition is sometimes seen in paralysed parts, and accompanying severe neuralgias.

Causes coming under the second head are

A. Causes acting within the artery itself, as:

a. Disease of the artery walls causing a thickening of the intima, or calcareous degeneration of the same.

b. The formation of clots on the vessel walls, impeding or entirely stopping the flow of blood.

B. Causes arising without the vessels which cause a narrowing of a limited portion of the artery, as:

a. Tumors.

b. Cicitrucial strictures

c. External constrictions, (bandages, ligatures, etc.)

d. Collections of fluid, or air in cavities having unyielding walls (effusions into ventricles of brain and pleuritic effusions).

Local anaemia may also result from a third cause, i.e. the presence of a pathological excess of blood in some portion of the body which draws off a certain amount of blood from a neighboring part.

RESULTS OF LOCAL ANAEMIA:

1. General results.
2. Effects upon the heart.
3. Effects on the vessels in the anaemic area.
4. Effects on the circulation in general.

1. Results of complete or partial occlusion of an artery depend upon the presence or absence of anastomoses in front of the seat of obstruction. If such exist the results are unimportant as the circulation is soon reestablished. If they do not exist we will have:

1. An increased arterial tension behind the obstruction.
2. A retarded stream beyond it.

The external effects noted in an anaemic area are paleness and coldness of the part.

If the obstruction is severe, but not occluding, there is a flow of blood into the anaemic area from the vessels

in the neighborhood, in which the pressure is higher than in the vessels supplied by the partly occluding artery. This blood joins the greatly diminished stream still flowing in the anaemic area and passes on through the capillaries leading from the part. The stream may be so sluggish that the heavier red blood corpuscles sink in it and are deposited in greater or less numbers in the vessels.

On the other hand where the obstruction is complete the blood pressure in front of the obstruction is zero while there is still a positive pressure in the surrounding vessels. The result is that a retrograde stream is established into the vessels in the anaemic area until the pressure within them equals that in the surrounding vessels. If this is high this retrograde stream may be so strong as to cause a veritable engorgement of the anaemic part, so that instead of appearing pale it is of a deep red color.

Tissue changes also occur in a part deprived of blood, but we will take these up later in the course

2. The effects of local anaemia upon the heart are important. If there is total occlusion of one of the coronary arteries, or of any considerable branch of one of them, there is, after the lapse of about 90 seconds a cessation of the heart beat. The heart stops in diastole and from this stoppage nothing can arouse the ventricles to renewed activity. In a certain number of cases of sudden death, the autopsy has disclosed, as the only apparent cause, the closure of a large branch of one of the coronary arteries. The condition known as angina pectoris, which is associated with intense pain radiating from the cardiac region, is found to be associated with advanced disease of the walls of the coronary arteries, by which their calibre is narrowed to a considerable extent, producing in all probability a partial anaemia of a portion of the heart. When <u>nitrite of amyl</u> is administered

by inhalation in this disease, there is an immediate cessation of pain. This is due to the fact that nitrite of amyl causes a wide dilitation of all the arteries, including those of the heart, and the local anaemia of that organ is relieved at least for the time being.

The paroxysms of pain in angina pectoris are prob. thus precipitated by a spasm of the arteries, thereby still more narrowing their already diminished calibre.

## 3. EFFECTS UPON THE VESSELS IN THE ANAEMIC AREA:-

If the anaemia has existed for a short time, say 1 to 2 or 3 hours, the first effect we will note on allowing the blood to flow through the vessels will be that they dilate. If the anaemia has lasted longer, say 8 to 10 hours, besides the dilitation of the vessels there will be a swelling of the part. This swelling is due to an escape of blood plasma, with a certain number of white blood corpuscles, from the vessels into the tissue. If the anaemia has lasted somewhat longer we will notice here and there small red areas in the tissues, which are due to an escape of the red corpuscles. The size and number of these haemorrhagic spots depends upon the dutation and severity of the anaemia. If the anaemia has lasted for 2, or more days the part commonly continues cold, dry and shrunken. The effect then of anaemia upon the vessels is to seriously injure their walls so that they no longer are capable of confining within themselves the blood plasma, or the blood cells. Their permiability has been made abnormal. This you must remember applies only to the veins and capillaries and not to the arteries.

If the artery that is occluded be a terminal artery, i.e. one that has no anastomosing branches beyond a certain point, the blood that forces its way into

the anaemic area, in the way described in the last lectures, would pass through the vessel walls in the way just described, and the meshes of the tissue of the part would become stuffed with blood, forming what is known as a <u>haemorrhagic infarct</u>.

## 4. EFFECTS UPON THE CIRCULATION IN GENERAL.

The question to be answered is when there is anaemia of any part what becomes of the blood that normally should go to that part?

In cases of anaemia due to any excess of blood in some other part, the answer is simple. In anaemias, however, due to the other causes already given the first effect noticed is a general rise in the arterial pressure, the pressure being as great in the artery just behind the obstruction as it is in the carotid artery or any other. This general rise of pressure continues until compensation takes place by decrease in the resistance to the circulation in some part of the vascular system.

Now we always find that this decrease in resistance occurs in that part where the blood is most needed and not, as some supposed, in the artery that first left the aorta above the artery that is occluded. For example, when there is anaemia of a part supplied by several arteries because of occlusion of one of them, we find that the amount of blood reaching the part through the remaining arteries is the same as before the occlusion of one of them. Take again the examples of anaemia of one kidney, for any reason, it is always found that instead of going anywhere in the vascular system, the blood that should go to the anaemic kidney goes to the other kidney and nowhere else. This is because the other kidney needs the extra supply of blood to enable it to get rid of the waste products that were formerly removed by both kidneys.

If for example we take the case of a leg from which the blood supply has been cut off, the blood does not go to the other leg, for that would not enable it to do the work of two, but not being wanted by any part it goes where it is most needed, that it is soon removed entirely, only so much blood remaining in the body, and being afterward produced as is nebessary for its nutrition.

# HYPERAEMIAS

By hyperaemia we mean the presence of more blood in a part than is normal. This condition may result from two entirely different sets of causes.

1. Those that determine an increase of blood flowing through the arteries of a part in a given time, or what we call active, or arterial hyperaemia, or active congestion.
2. Those that determine an overfullness of the veins leading away from a part, or what we call passive, or venous hyperaemia, or passive congestion.

## ACTIVE HYPERAEMIA

CAUSES:

1. Severe anaemia of an organ will as we have just seen in the case of the kidney, cause a hyperaemia in the corresponding organ of like function. These are called secondary, or collateral hyperaemias.
2. All other active hyperaemias depend upon some abnormal diminution of the resistance to the arterial flow.

Such a condition may result from causes which:

a. Stimulate the muscular coats of the arteries to actively dilate; as heat, electricity, friction and the like.

b. From causes which paralyze the muscular coats of the arteries allowing them to dilate because of internal pressure; as cold, where there is first a local anaemia which is quickly replaced by hyperaemia, a blow, or the removal of great pressure from the arteries, as the removal of a tumor, or the drawing off of an extensive ascites.

c. From causes which stimulate the vaso-dilator nerves supplying the arteries, as wounds of the brachial plexus which are followed by the condition known as "glassy-fingers," and which is an active hyperaemia of those parts. These hyperaemias are known as neurotonic.

d. From causes which inhibit, or paralyse the vaso-constrictor nerves supplying the arteries, thereby removing the normal arterial tone, and allowing them to dilate because of internal pressure. Such hyperaemias are known as neuroparalytic.

Both the neurotonic and neuroparalytic hyperaemias may be set up by diseases in organs distant from those in which the hyperaemia occurs, as for example the hyperaemia which results in the kidney when there is disease of the ovaries. These we speak of as reflex neurotonic or neuroparalytic hyperaemias.

Disease of the central nervous system, the brain or spinal cord, may also set up either a neurotonic, or neuroparalytic hyperaemia by affecting the centres from which these nerves arise.

RESULTS OF ACTIVE HYPERAEMIA: Provided the condition is uncomplicated the results are unimportant, except in those cases where the hyperaemia is followed by an increased growth of the part. This occurs for example in the kidney where on account of the removal or anaemia of its fellow there is need of increased functional activity to remove the waste products from the system. Why such an hypertrophy should not always occur is not clearly understood

# PASSIVE HYPERAEMIA.

SYNONYMS:- Passive Congestion, Venous Congestion.

We have seen that it is possible for the arterial system to become over-charged with blood from various causes, producing what is spoken of as Active Hyperaemia. As we follow the course of the blood stream it next enters the capillary system and the question which presents itself is, - can the resistance in these vessels undergo such changes as to influence the general circulation? Since these minute vessels are not supplied with any nervous or muscular mechanism such as is found in the larger vessels, they are therefore unable to actively dilate or contract, and can therefore not materially affect the general circulation in the veins and arteries. Should any external resistance be interpolated in the capillary stream its effect would be practically of no account because of the extensive system of capillary anastomoses.

When we come however to the veins which are next in order as we follow the circulation, we find that, as in the arterial system, certain important changes can take place. First, in order to understand these changes we must note the differences which exist between the structure of the veins and that of the arteries. In the first place the muscle coat of the veins is less highly developed than it is in the arteries, and in the second place the veins are not supplied with any nervous mechanism such as is found in the arteries and which allows them to actively dilate and contract. These facts mean that there is nothing in the venous system which we can compare to what we call arterial tonus, and that we can not have a venous hyperaemia, as we have an arterial, due simply to the falling off of venous resistance. In other words in order to have a venous congestion there must be some external resistance offered to the efflux of blood from the veins.

## HYPOSTATIC CONGESTION AND SIMPLE PASSIVE HYPERAEMIA:-

Among passive congestions we may recognize two varieties, one of which, simple passive congestion, is dependent upon some simple hindrance to the outflow of blood from a part, and the other hypostatic congestion, dependent upon a more complicated interference with the mechanism of the circulation.

The forces which maintain the flow of blood against the action of gravity are (1) The heart's action, (2) the action of the skeletal muscles in conjunction with the valves of the veins; and (3) the suction action of the thorax, the so-called aspiration of the thorax. Now, if any one or more of these forces be seriously interfered with the blood will circulate less freely than normal and will tend to settle in the most dependent portions of the body. This form of congestion is what is termed hypostatic congestion, and the most typical examples of it are to be found in cases of continued fevers, such as typhoid. Here not only is there cessation of all muscular movements, but both the heart's action and the respiration are enfeebled, consequently we find that in such dependent parts as the bases of the lungs, the calves of the legs, the buttox, and the undersurfaces of the various organs, the blood has settled until all the vessels are greatly over-distended, producing a marked grade of congestion.

All other passive congestions, as has been stated, are the result of some simple interference with the outflow of blood from the veins of a part, such as tumors, bandages, etc. pressing upon them.

RESULTS:- Whatever the cause of the congestion may be, the results are always the same and may be considered under the following heads.

1. Effects upon the circulation in the veins
2. Effects upon the vessels in the congested area.
3. Effects upon the general circulation
4. Effects upon the tissues in the congested area.

I. The effects upon the circulation in the veins can be very simply stated for the blood behind the point of interference will flow more slowly than normal, or will come to a standstill if the occlusion of the vessels be complete, and the flow beyond the point of interference will be diminished or stopped altogether

II. The effects upon the vessels in which the congestion occurs are, however more complicated. We can best study these under the microscope, after we have produced a congestion by tying a vein in the web of a frog's foot. When this has been done the following changes will be noted

1. There will be a decrease in the velocity of the stream depending upon the amount of occlusion.
2. As the current becomes slower and slower a certain amount of dilitation of the vessels will be observed
3. The congestion becomes apparent first in the larger vessels and then passes gradually backward into the smaller veins and finally into the capillaries.
4. After a time as the vessels become fuller the peripheral zone of clear plasma is encroached upon and finally disappears, the red corpuscles completly filling the lumen of the vein.
5. Finally the circulation becomes extremely sluggish, or may even progress as in the arteries with a pulsating motion and the vessels appear as solid red rods in which for moments at a time no motion can be seen.
6. As we watch these vessels we can see in places small bud like projections along their outside. In time it is seen that these are red corpuscles which are passing directly through the unruptured vessel walls. This process is spoken of as the <u>diapedisis</u> of the red corpuscles.

7. In addition to these changes there is also an escape of the fluid portion of the blood through the vessel walls. This escape causes a certain amount of swelling in the congested area. It is important to note that this fluid which is expressed because of the increased pressure within the vessels differs materially from the plasma of the blood and also from normal lymph in that it is poorer in albumen, and has very little power of coagulating. There has been therefore some subtle change produced in it by its passage through the vessel wall.

III. The effects upon the circulation in general are worthy of note in that they differ materially from the effects of a local anaemia. We saw that when an artery was occluded there was a general rise in arterial tension throughout the entire arterial system for a time. When however there is occlusion of a vein, the increase in pressure is confined to the vessel occluded and to its immediate venous and capillary tributaries, this increase never being transmitted to the arteries which supply the occluded vein. Within the area of congestion this venous pressure may be very great and may even equal the pressure in the arteries but it never exceeds it. This is probably to be explained by the fact that the anastomoses in the venous and capillary systems being much freer than in the arterial, the blood is carried off by side channels and so a higher pressure is prevented.

(A part which is in a state of passive congestion will appear swollen, (Why)? redder than normal, (why) and will be reduced in temperature, (why)?

Passive congestions are most frequently caused by valvular lesions of the heart, such lesions acting to produce an anaemia of the arterial system and a congestion of the venous. (Explain the production of passive congestion of the liver by mitral regurgitation. By Aortic stenosis).

# THROMBOSIS.

## — CLOTTING THE BLOOD. —

Before proceeding to the study of the next pathological process, i.e. the clotting of blood within the vessels, let us review the process of clotting as it takes place when the blood is drawn.

Blood drawn from the body quickly becomes converted into a solid mass, the clot which after a short time shrinks somewhat, and as it does so squeezes from itself a clear straw colored fluid, the serum in which it floats. We thus have gotten from blood two products, the clot and the fluid which surrounds it, which we call serum.

On careful examination we find that the clot is composed of the corpuscles of the blood bound up in the meshes of a tough fibrous, elastic material. This material is the essential constituent of the clot and is called fibrin, the corpuscles are present only because when the fibrin was formed they were present and became entangled in its meshes.

Besides blood, there are certain fluids occasionally found in the body, such as hydrocele fluid, which though they do not clot spontaneously can be made to clot upon the addition of a little blood, blood clot, or serum, and on clotting yield normal fibrin.

Now it is found that the substance in blood, blood clot and serum, which causes clotting and which can be isolated from these substances, belongs to the class of bodies known as ferments, or substances which by their presence cause the formation of new products into whose composition, however, they do not enter. These bodies are called ferments, and this particular substance therefore fibrin ferment. A very small amount of this ferment is able to set up extensive clotting.

It is found also that it is necessary to use the entire hydrocele fluid to get clotting on the addition of a little

fibrin ferment, but that a substance can be isolated from it which in the presence of fibrin ferment produces a perfect clot, while the hydrocele fluid from which it has been removed will not clot even if large amounts of fibrin ferment is added to it. This substance, from its intimate connection with the formation of fibrin has been called fibrinogen.

We may say, then, that clotting or the formation of fibrin, results from the action of fibrin ferment upon a substance, found in blood hydrocele fluids, etc., which is known as fibrinogen.

The question as to the origin of fibrin ferment is yet to be definitely settled, but for the present we may say that we believe it to be derived in some way from the breaking down of the white corpuscles of the blood, and possibly from the same process in other cells of the body.

According to this we would see why it is that hydrocele fluid, which contains no white corpuscles, does not clot spontaneously and does clot upon the addition of blood, blood clot and serum, which has been formed in the process of clotting amust therefore contain the products of the destruction of white, as well as of red corpuscles.

## THROMBOSIS.

Thrombosis is the term applied to the clotting of blood within the living vessels. The process is essentially the same as that we have just described.

### CAUSES OF THROMBOSIS.

So long as the blood is in motion within healthy vessels it remains fluid, but we have seen that as soon as it is drawn it begins to clot. We are justified therefore in assuming that both its motion, and contact with healthy walls are the chief factors in maintaining its fluidity. Serious disturbance of either of these conditions will result in clotting of the

blood in the vessels. We may, then, that the formation of a thrombus is the result primarily of some injury to the vessel walls, for it is probable that slowing of the circulation beyond a certain point induces thrombosis chiefly because the vessel walls are injured by not being supplied with sufficient nourishment by the sluggish flow of blood. Changes which effect the lining endothelium of the vessels therefore will tend to produce thrombosis. Such are for example:

Inflammation.
Atheroma and atheromatous ulcers.
New growths growing into the vessels
Compression of the vessels, etc., etc.

Retardation of the flow as I have just said powerfully predisposes to the formation of thrombi. In this way are explained the thrombi formed in aneurisms, in the veins of the pelvis during pregnancy, in the appendix of the right auricle and apex of the right ventricle in conditions of weakened circulation. These are called marantic thrombi. They are also found to occur during the course of all wasting diseases, especially phthisis and cancer in which there is great debility and enfeebled heart action.

For some time these thrombi were thought to be formed simply because of slowing of the circulation, and to be unaccompanied by any observable morbid condition of the vascular endothelium, but careful microscopic examination will show that the endothelium is absent or defective at the seat of these thrombi. Whether these defects in the endothelium preceded the formation of the thrombus or were caused by it, it is hard to determine.

After severe fevers, such as typhoid, extensive thrombi are sometimes formed. These may probably be classed under marantic thrombi and considered as due to enfeebled circulation and the vascular changes that it produces. In such conditions also there is poor nutrition, and the inner coats of the vessels are especially liable to suffer. Micro-

organisms may also be present which probably have some effect in favoring coagulation.

As has been said, cancer and tumors growing into the vessels are occasional causes of thrombosis. At times there is found an extension of the cancerous growth into the thrombus and when portions of this are washed away in the circulation as frequently happens, we have where the portion lodges the foci for a new cancerous growth.

Again, when a thrombus is caused by inflammation of the vessel or of the area through which it passes, it may become infected with whatever specific poison caused the inflammation, or is found in the pus in an area of suppuration. If this takes place and portions of the thrombus become washed away, as in the case of cancer, there are numerous foci of the disease started wherever these portions of the thrombus lodge. In this way the general disease pyaemia is caused.

A thrombus once formed acts as a foreign body in the circulation, and has a tendency to extend in both directions. It usually extends at least from the point of origin to the nearest branch given off from the vessel. It may extend even farther and involve vessels at some distance from the seat of the original thrombus.

## ANTE AND POST-MORTEM CLOTS.

Clots formed in the heart and vessels after death differ materially from the thrombi, or clots formed in the vessels before death. Post mortem clots are dark red, almost blackish, shiny masses, very soft and juicy, and frequently found at autopsies occupying the cavities of the heart, and portions of the larger vessels. They are easily distinguished from the clots formed before death by the fact that they are darker in color, and are never adherent to the walls of the vessels, or heart; they are also more soft and easily torn.

# STRUCTURE OF WHITE THROMBI.

The mode of formation of the white thrombi has long been a matter of dispute, some claiming that they were in the earliest stages due to the collection at the seat of injury, of numerous leucocytes, which underwent degeneration and so gave rise to fibrin, while others held that these thrombi were not coagula at all, but were due to an accumulation of blood plates at the seat of injury, which underwent a sticky metamorphosis, forming therefore a mass by conglutination, not by coagulation.

Dr. Welch of the Johns Hopkins University undertook a series of experiments to try to determine what was the true part played by both these elements, the blood plates and the leucocytes in the early stages of the formation of these thrombi. The results of his work are briefly as follows:

In the very earliest stage, say within five minutes after injury, there is at the seat of injury a collection of blood plates which presents the sticky change described by some observers. There is at this time no fibrin, and but very few, if any, leucocytes. A short time later, however, sections of the developing thrombus shows a larger number of leucocytes and some fibrin, and as the thrombus grows older there is an increasing number of leucocytes and an increasing amount of fibrin, so that at the end of a half hour or so the clot consists of a mass of blood plates intimately attached to the wall of the vessel in which there are numbers of leucocytes and a considerable amount of fibrin, so that although these two latter elements are absent from the very earliest stages, they must be considered as essential elements in the structure of the completed thrombus. It also follows that though the earliest stage is one of the conglutination of the blood plates, that the thrombus is in reality when completed, not a conglutination, but a true coagulation, as fibrin is present in considerable amount. The reason that fibrin is absent in the very earliest stages is probably because there are at this time no leucocytes present to give rise to fibrin by the formation of

the necessary fibrin ferment. Thrombi are spoken of as parietal and occluding the terms being self explanatory.

## LATER CHANGES IN THROMBI.

Red thrombi at first resembles ordinary blood clot formed outside the body, but as they grow older they gradually contract so that they become dryer and firmer. As it undergoes these changes the walls of the vessel also shrink somewhat so that the thrombus becomes attached throughout its length to the walls of the vessel. There then follows a breaking up of the red corpuscles and an absorption of the coloring matter, the thrombus becoming gradually paler and paler, so that the color of one of these bodies is an index to its age. Following this condition any one of the several changes may take place, of which these are the most common,

Organization
Calcification
Softening

ORGANIZATION.- When this takes place there is a formation within the clot of new connective tissue, derived either from the leucocytes which wander into the thrombus from the vasa vasorum of the injured vessel, or from the endothelial cells of the vessel, or from both, (the question is still in dispute) which ultimately converts the thrombus into a mass of this tissue adherent to the vessel walls and making the vessel in time look like a fibrous cord. In this process the clot itself takes no active part, the cells simply grow in it and it itself is gradually removed. Occasionally, by the contractions of this newly formed connective tissue, channels are made through its mass so that ultimately the blood again flows in practically its old channel. This latter process is spoken of as canalization of the thrombus.

CALCIFICATION.- Occasionally occurs, especially in the clots formed in varices, giving rise to little form

calcarious masses known as phleboliths.

## SOFTENING.

Is of two kinds:

1. Simple or bland softening.
2. Septic softening.

In simple softening the mass begins to break down in its centre into a pus like greyish material composed of fatty and albuminous granules, fatty degenerated leucocytes and blood pigment. This continues till the whole thrombus is softened. Portions of this mass may be washed away in the circulation.

Septic softening is far more serious in its results. The process is essentially the same, but here the softened mass contains bacteria, chiefly septic micrococci. Portions of this softened septic mass entering the circulation are scattered throughout the body forming wherever they lodge abcesses and often giving rise to the most serious results.

## RESULTS OF THROMBOSIS.

The results of thrombosis are:

1. Changes in the walls of the vessels.
2. More or less obstruction to the circulation
3. Embolism.

1. When a thrombus becomes organized the walls are at first thickened and infiltrated everywhere with numerous small round cells, but ultimately with the thrombus they atrophy. It is when a thrombus undergoes a process of septic softening that the most important changes of an acute inflammatory nature takes place in the vessel. These changes are due to the irritation of the decomposing thrombus, and are most frequently observed in veins. The wall of a vein within which a thrombus is undergoing septic softening is considerably thickened and resembles more the wall of an artery. The inner surface

loses its transparency and is of dull opaque color. The coats are all injected and present numerous hemorrhagic spots which are often visible through the intima. The swelling of the walls is due to the dense infiltration with leucocytes which conceals the normal structure. The innermost cells die and are shed into the lumen of the vessel. Small collections of pus may be seen in the middle and external coats.

This acute inflammatory condition of the veins constitutes what is known as suppurative phlebitis.

2. Obstruction to the circulation depends in its results of course upon the size of the vessel occluded, the presence or absence of collateral branches, the rapidity of the formation of the thrombus, and upon the force of the circulation. There will be produced as the case may be local anaemia or passive congestion with the results which we have considered in studying these conditions.

3. Portions of a thrombus may be washed away to lodge ultimately in some other parts of the circulation, giving rise to the condition known as embolism which we will consider later.

# EMBOLISM.

An embolus is a plug of some nature transported by the blood current from one situation to another. The term embolism is applied to the process of obstruction of a blood vessel by an embolus and to the disturbances resulting therefrom.

## SUBSTANCES THAT MAY FORM EMBOLI.

Substances that may form emboli are any foreign bodies that find their way into the circulation, such as: fragments of thrombi, small masses of bacteria, bits of tumor, fragments of diseased cardiac valves, clumps of pigment, oil globules and bubbles of air.

An embolus cannot be arrested in the veinous system, except in the portal veins, or in the cavities of the heart, because the blood current is from smaller to larger vessels. It is in the arteries, therefore, that we find emboli, except in very rare cases. Obstruction of an artery that is well supplied with anastomosing branches is without noticeable result, provided the embolus be what is termed "bland"; if however the artery be without anastomosing branches beyond the point where the embolus lodges, or in other words, if the artery be a terminal one, we have as a result of the embolism, the formation of an infarct. Beyond the embolus, the tissue deprived of its blood undergoes what is known as coagulation necrosis, the protoplasm of the cells dies and goes over to a substance closely resembling fibrin, hence the term "coagulation". The essential thing about an infarct is this necrosis of the part. Infarctions may be either hemorrhagic, or anaemic, the latter being sometimes spoken of as "white infarcts". Why some infarcts are hemorrhagic and some anaemic it is difficult to explain, but the varying density of the tissues probably is an important factor; thus in the lung, where the tissue is open

and lax we always get hemorrhagic infarcts, while in the kidney, whose tissue is dense and compact, we always get anaemic infarcts. The method of formation of an hemorrhagic infarct has already been given. When an embolus lodges in an artery and occludes it we will have a local anaemia of the parts supplied by the artery beyond the point of occlusion.

Emboli consisting of portions of infective thrombi will produce at the points where they lodge such perious results as abcess formation and extensive necrosis of tissue. It is in this way that pyaemia is sometimes produced.

The source of fat emboli is to be found chiefly in fractures of the bones and laceration of the marrow with rupture of vessels large enough to allow the entrance of particles of fat, which are then transported to various parts of the body, and are frequently found in large numbers in the capillaries of the lung. Fat embolism, though quite extensive at times, is comparatively harmless.

## AIR EMBOLI.

Air emboli on the other hand are frequently fatal, provided sufficient air enters the circulation

Air usually finds entrance to the circulation through incisions of large vessels near the heart. When this occurs the fatal termination is to be attributed to the accumulation of a considerable amount of air in the cardiac cavities, the contractions of which are unable to force the elastic air forward. The air remaining in the right auricle and ventricle is an obstacle to the entrance of blood from the venae cavae and arrests the pulmonary and systemic circulations.

Emboli can set up thromboais, and the thrombi thus formed do not differ from the thrombi which we have studied, and undergo all the changes possible to the latter.

# INFLAMMATION.

## DEFINITION.

Inflammation has been variously, defined ever since the time of Celsus (Third century), but no definition short of a description of the various phenomena presented by an inflamed tissue will give us any adequate idea of the process. Celsus described a part as inflamed when it was red, swollen, hot and painful. To these so-called four cardinal signs of inflammation, recent writers have added a fifth: loss of function of the inflamed part

As it was well known that an injury severe enough to cause an immediate death of a part could not set up inflammation, Sanderson has defined this process as "the succession of changes which takes place in a living tissue as the result of some kind of injury, provided this injury be insufficient immediately to destroy its vitality." In order to give an intelligent definition of inflammation we must be able to describe the various changes which take place in a tissue which is inflamed

There have been two views held with respect to the process of inflammation. According to the one, the essential phenomena of inflammation are referable to the blood and blood vessels. According to the other the essential changes occur in the solid tissues surrounding the vessels, and these are only secondarily affected. Virchow, by his work, gave great stimulus to this latter view, but when Cohnheim discovered the power of the leucocytes to wander from the vessels and made a study of the vascular phenomena of inflammation he established the truth of the former. According to Virchow and his pupils the great number of small cells, of embryonic, appearance, seen in a tissue that is inflamed, are derived from the proliferation of the fixed tissue cells of the part. According to the researches of Cohnheim these cells are largely, though not entirely derived from

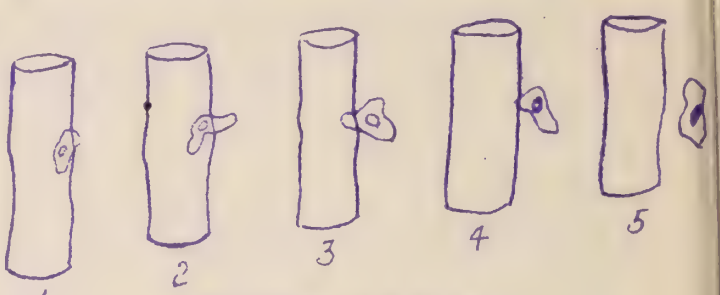
Inflammation is essentially a local reaction

the blood, by the migration of the white corpuscles.

## THE VASCULAR PHENOMENA OF INFLAMMATION.

1. Almost immediately after the part in which the changes are to be studied, is injured, there is seen to be a momentary contraction of the vessels, after which
2. There is seen to be a gradual dilatation beginning with the arteries which are the most affected and extending to the veins which are less, and to the capillaries, which are the least affected. This dilatation may continue for 10 to 12 hours. At the same time that the vessels dilate, there is for a short time (an hour or so) an increase in the rapidity of the blood stream. We have therefore an active hyperaemia of the part.
3. The next thing noticed as we watch the advancing inflammation is that the rapidity of the blood stream diminishes until in places there is almost stasis. Coincident with this slowing of the stream one sees a peripheral arrangement of the white blood corpuscles growing gradually more and more distinct while the vessels become more and more engorged with blood. The slowing of the circulation probably depends in part upon this accumulation of leucocytes in the vessels, and also upon molecular changes in the walls of the same producing more friction from the passing corpuscles, and lastly upon the fact that the tissues of the part surrounding the capillaries, as well as the walls of the vessels, loose a certain amount of their elasticity and are unable to act toward the column of blood as the healthy walls and healthy tissue formerly did.
4. After a time if we carefully watch the vessels in an inflamed area we will see that here and there along the outside of the capillaries and smallest veins (but never arteries) small buds appear, and that

opposite each bud, within the vessel there is the body of a white blood corpuscle. As we watch this bud we will see that it grows larger and that at the same time the body of the leucocyte within the vessel grows smaller. Before long the leucocyte will be pseudopoing free without the vessel This process of the escape of the leucocytes from the vessels, which is very characteristic of inflammation, is spoken of as the migration of leucocytes By this means the tissues in an inflamed area are often filled with thousands of these little wandering cells, which afterward have important parts to play.




Figures 1, 2, 3, 4, 5.
Illustrating of migration of Leucocytes.

5. While the leucocytes are thus escaping by their own powers of migration, the red corpuscles are also escaping by the process of diapedesis which as we have already seen is purely a passive one. The severer the inflammation, the larger will be the number of red corpuscles which escape into the tissues in this way.

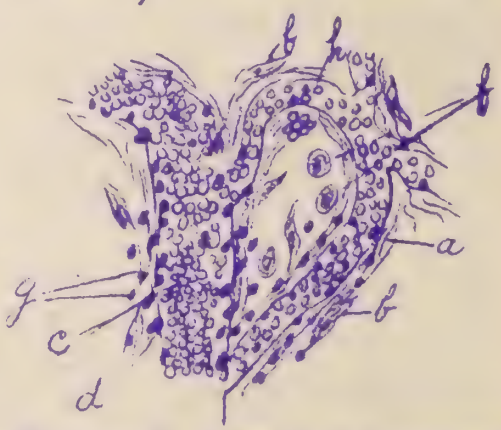



Figure 6. Vessels from inflamed Omentum
a. Normal fibrous trabeculae.
b. Normal Endothelium.
c. Vein with White Corpuscles in peripheral Stream
d. Emigrating White Corpuscle
f. Desquamated Endothelium
g. Emigrated White Corpuscle.
h. Escaped Red Corpuscles.

6. While the migration of the white corpuscles and the diapesis of the red ones is going on there is also an escape of fluid from the vessels. This fluid differs from the fluid which escapes as we have seen in passive con-

gestions, in being more nearly like the normal plasma of the blood, that is in having more albumen and salts, and in possessing the power of clotting to a greater degree. The more severe an inflammation is the more nearly does this fluid which escapes resemble blood plasma, while the milder the inflammation is the more nearly it resembles the fluid of oedema that is found in cases of passive congestion

## THE CAUSES OF INFLAMMATION.

The causes of such alterations in the circulation as we have just described as being characteristic of inflammation, must therefore be the causes of inflammation. The number of agents that can act in this way is very large and we can say as a general rule that mechanical, thermal and chemical injuries alter the vessels in this way and produce inflammation.

## LEUCOCYTES AS CARRIERS.

After the leucocytes have escaped from the vessels and have wandered away into the tissues, they play an important part frequently as carriers, or scavengers, as they have sometimes been called. They carry away injurious substances or the products of the destruction of tissue with which the parts are encumbered. These materials carried off may be fat globules as in the case of destruction of nerve tissue, pigment granules as in the case of the destruction of red blood corpuscles, particles of carbon, etc.

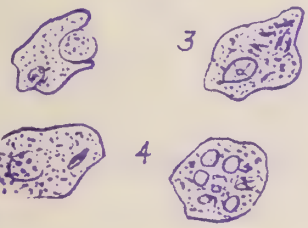



Fig 7.

Leucocytes as Carriers.

1. Leucocytes surrounding a corpuscle containing Malarial parasite.
2. Leucocyte with Malarial parasite within its protoplasm.
3. Leucocyte containing bacteria
4. " " Oil droplets

A more especial importance belongs to this property of the leucocytes, when they attack and attempt to remove

injurious micro-organisms. That they do this has been abundantly proven, as they have been found with the micro-organisms within their protoplasm. It is no uncommon sight to see leucocytes which have devoured the plasmodium of malaria. In this struggle with the injurious micro-organism the leucocytes are not always successful and when not so is killed and has to be removed at some other time and in some other way. When, however the leucocytes are successful they help the organism to overcome the injury that has set up the inflammation. As a further proof that the power of amoeboid movement of the leucocytes, by which they are enabled to act as carriers is of importance in the process of inflammation we have the fact that in certain diseases where this power is impaired or abolished, inflammation and repair are very imperfectly performed, if performed at all. Such a disease is leukemia.

Leucocytes which have retained their vitality in this struggle with harmful agents may re-enter the circulation either by passing directly through the vessel walls again, or by way of the lymphatics. It is probable that this latter route is the more frequent. Those leucocytes which perish, go with other cells to form pus and are subsequently removed if the part recovers. It is probable also that some of these wandering cells subsequently develope into connective tissue cells to help repair any injury that may have been done, though most of the connective tissue that is found to replace tissues lost, is developed from pre-existing connective tissue cells.

## PUS.

Pus which is formed in the course of an inflammation is composed of the bodies of dead leucocytes, and also of cells which have been proliferated by the fixed tissue cells of the part, which when first formed can not be

distinguished from the small round leucocytes. These cells may come either from muscle, connective tissue, or other tissue cells, and when young they exhibit the same amoeboid movement as do the leucocytes.

## TISSUE CHANGES IN INFLAMMATION.

Not only as we have just seen do the tissue cells of the part proliferate and produce young cells which at first can in no way be distinguished from leucocytes, but there is also going on a certain amount of tissue destruction. The cells injured by the process atrophy, degenerate, disintegrate and are absorbed or removed. The new cells that are formed are however, the elements that finally bring about the repair of the part. There is therefore going on at the same time, side by side, tissue destruction and tissue repair. The chief factor in this repair is the increased supply of nourishment to the tissues, which increased supply is furnished by the vascular changes that take place and which have been described. We are justified therefore in speaking of the vascular changes at least as conservative and tending to the repair of the part. If we could imagine a tissue inflamed or [illegible] these vascular changes did not take place, and in which therefore the tissues were not supplied with increased nourishment we would have instead of repair, necrosis or death. Also the destruction of a certain number of cell elements, removes a certain amount of pressure from those cells that are left, and allows them more room in which to grow and multiply.

## REPAIR OF CONNECTIVE TISSUE.

This has an especially important relation to inflammation because (1) being present in all organs the phenomena of its restoration is always seen in inflamed

parts. (2) It has a greater power of repair than any other tissue and therefore frequently takes the place of other tissues when they are imperfectly repaired. (3) The process of its repair sometimes passes necessary limits and an excess is produced which is frequently injurious. This formation of an excess of connective tissue is spoken of as an hyper-plasia of connective tissue

## GRANULATION TISSUE.

One of the essential steps in the process of repair of connective tissue is the effusion of a coagulable exudation. Formerly it was thought that the fibrin of this exudate was directly converted into connective tissue This we now know not to be so. The fibrin of the exudate plays only a passive part and the essential change consists in the substitution for this, of true tissue formed [illegible] pre-existing connective tissue cells, and possibly from the migratory cells (leucocytes) to a certain extent. This tissue is then supplied with vessels that grow out from the vessels already existing in the neighborhood. The result of such a process is the formation of a tender temporary tissue that ultimately becomes true connective tissue, and the term given to this temporary structure is granulation tissue. Such temporary tissue is seen filling the cavity formed by deep ulcers and in the healing of nearly every kind of wound. Not all the leucocytes that escape from the vessels go to form new connective tissue. As has been said large numbers perish and enter into the formation of pus. Others, according to Zeigler develope into large granular cells which he has called fibroblasts which become elongated, spindle shaped, or branched, and ultimately develope into connective tissue fibers, either directly or give rise to connective tissue fibers by the fibrillation of the inter-cellular substance. Those who deny Zeigler's explanation of the origin

of the fibro-blasts from leucocytes, look upon them as derived from the fixed connective tissue cells of the part. It is probable, however, that both processes take place.

The new blood vessels found in granulation tissue are produced by solid protoplasmic processes from the vessels in the neighborhood. These processes ultimately become hollow, and have developed about them the walls of veins or arteries.

Such new formed tissue and blood vessels as have just been described are formed in wounds and ulcers that are healing, and also in the development of adhesions after inflammation of serous membranes, the pleura, peritoneum and the like.

New formed connective tissue of this kind is also seen in the case of Chronic inflammation of solid organs such as the liver and kidney and its production is frequently in excess of that already existing, and constitutes what is called an hyper-plasia of connective tissue as already mentioned. This new formed tissue has always a marked tendency to contract, and in this way is explained the contraction of superficial scars as well as the lobular appearance and decrease in size of those internal organs in which there has been a Chronic interstitial inflammation. This contraction in the latter cases produces such pressure upon the true cells of the organ that they frequently atrophy.

The formation of granulation tissue must be looked upon as a step in the normal process of repair. It sometimes happens, however, that such tissue, though formed shows no tendency to go on to the completion of the process in the formation of true connective tissue. This takes place in such diseases as tuberculosis and syphilis. In the case of tuberculosis of the skin (lupus) for example there are masses of this tissue formed which though it looks as though it would heal never does. There remains a condition of permanent granulation tissue showing no

tendency to go on to the formation of normal connective tissue.

## INFLAMMATIONS MAY BE CLASSIFIED:

I. According to the character of their exudate
II. According to the tissue affected.
III. According to the rapidity of the course they run.

I. According to the character of the exudate we have among others the following

A. Fibrinous.
B. Serous.
C. Purulent.
D. Hemorrhagic.

### Fibrinous.

Those, the exudate of which contains in addition to the cellular elements, and fluid transuded from the blood, a considerable amount of fibrin. The best illustrations of this condition are fibrinous inflammations of serous surfaces, acute pleurisy, peritonitis, etc. A fibrinous exudate may also accompany inflammations of solid organs. In the lungs in croupous pneumonia we have a fibrinous exudate. Fibrinous inflammations of mucous membranes are called either diphtheretic, or croupous. Diphtheretic if the injury and consequent death of the tissue has been enough to involve not only the epithelium, but the tissue below it. In this case the fibrin of the exudate extends into the necrotic tissue, and the membrane formed by the inflammatory exudate can not be removed without some loss of tissue. Croupous if the injury has only produced a death of the epithelial cells of the mucous membrane. In this case the fibrinous exudate formed rests only on the surface of the membrane and can be readily removed without any loss of tissue. The terms diphtheretic and croupous are here used in purely an anatomical sense, and have nothing to do with the diseases diphtheria and croup.

## Serous.

Those, the exudate of which bears a strong resemblance to the transudations of passive congestion. The fluid of the exudate is poor in albumen and possesses little if any power of clotting and so contains little if any fibrin. Serous inflammations when involving mucous membranes are spoken of as catarrhal and in these cases the exudate contains no fibrin, but is made up of serum mucous and cellular elements.

## Purulent or Suppurative.

Those, the exudate of which is characterized by the presence of pus-cells in such quantity as to render it opaque and creamy.

The production of a purulent exudate depends upon the following:

1. An inflammation of a certain degree of intensity.
2. Acting for a sufficient length of time, and whose
3. Cause has the property of preventing the formation of fibrin.

No mild irritant acting for a long time, or powerful irritant acting only for a short time would cause suppuration. The essential element therefore in the production of a suppurative inflammation is the production by the exciting cause of the property of preventing the formation of fibrin, for should the exudate coagulate we would have a fibrinous, and not a suppurative inflammation.

The causes of suppurative inflammation are micro-organisms known as pyogenic micro-organisms from their power of producing pus. The most common are the staphylococcus pyogenes aureus, the staphylococcus pyogenes albus, and the streptococcus pyogenes. The gonnococcus is also a pus producing organism.

II. According to the tissue affected we have the following classes:

A. Parenchymatous
B. Interstitial.

Parenchymatous

Inflammations affecting the proper tissue cells of an organ, its parenchyma as it is called, is spoken of as a parenchymatous inflammation, it is chiefly marked by a more granular appearance of the cells than normal, the cells also staining poorly and appearing somewhat swollen. This condition of parenchymatous inflammation is sometimes spoken of as "cloudy swelling" when found in solid organs such as the liver and kidney. Inflammation affecting the air vesicles of the lung is parenchymatous, and constitutes the different forms of pneumonia.

Interstitial.

Inflammation affecting the connective tissue structure of a part, or organ is spoken of as interstitial. It is characterized by an hyperplasia of connective tissue, especially if the inflammation be chronic. If acute there seems to be a tendency to abcess formation. The chronic hyperplasia of connective tissue frequently results in secondary damage to the parenchymatous cells on account of the pressure exerted upon them by the contraction of the newly formed connective tissue.

III. According to the rapidity of the course they run we have:

A. Acute.
B. Chronic.

An Acute inflammation is one which runs its course rapidly and then declines. The word acute meaning rapid, not severe

A Chronic inflammation is of course just the opposite of the above. They may come on as acute inflammations subsiding and becoming slow in their progress,

or their onset may be insidious and their course chronic from the first.

## THE TERMINATIONS OF INFLAMMATION MAY BE EITHER.

1. Resolution.
2. Necrosis
3. New growth.

### Resolution.

This is the most common result and the most favorable. For it to take place the vessels must be restored to their normal condition so that no more exudation takes place; the exudate present must be removed, and the cause of the inflammation must cease to act.

### Necrosis

Will be described later.

### New Growth.

Especially noted when the inflammation becomes chronic and we have an hyperplasia of connective tissue.

# The Infective Granulomata

In certain diseases there is formed a tissue which so closely resembles granulation tissue in its structure that it has given to these diseases the name of granulomata. It has been further found that all of these diseases are, what are termed infective, that is they are all locally invasive, spreading from one focus of disease in all directions into the surrounding tissues; they are also able to infect distant parts of the organism by way of the blood and the lymphatic system; and finally they can be transmitted from one individual to another. For these reasons these diseases characterized by tissue closely resembling granulation tissue, and which are infective, have been called the "infective granulomata".

Granulation tissue, as you will remember is a temporary tissue in which is ultimately developed new connective tissue. It consists of masses of small round embryonic cells, each having a large nucleus which stains well, and little protoplasm. These are spoken of as the lymphoid cells, and are derived in large measure from the blood, and also from the proliferation of pre-existing connective tissue cells. Among these cells there are seen after a time large cells with nuclei that more closely resemble those of epithelial cells, from which they have been called epithelioid cells. These cells are the ones which give rise to connective tissue,

FIGURE 8. Forms of cells found in granulation tissue, and in the infective granulomata.
1. Lymphoid cells.
2. Epithelial cells, or fibroblasts
3. Giant cells.
4 Fibroblasts developing into connective tissue
(Modified from Zeigler).

which has led to their being called also fibroblasts. In normal granulation tissue we also see at times a very few giant cells.

In the granulomatous diseases there is formed a tissue which though it closely resembles granulation tissue, never developes beyond a certain point. It developes as far as though the formation of fibroblasts, and there seems to come a stop. After a time this tissue begins to degenerate.

Tuberculosis is the typical example of this class of diseases and the characteristic structure of tuberculosis is the tubercle. In its earliest stage a tubercle is simply a mass of small round lymphoid cells which infiltrate the tissue at the point of irritation by the tubercle bacilli. In the liver where it is most easily studied, these beginning tubercles are very readily made out. The mass of cells assume a nodular arrangement, either round or oval and at first consists only of lymphoid cells. It grows to a certain size and then we notice that the cells of the tissue in which it is developing have begun to die, their nuclei stain poorly, and are fragmented. Closely studying the cells of the tubercle we see two larger cells such as we saw in normal granulations, the epithelial cells. Staining the mass for bacteria we find in it the tubercle bacilli. Somewhat later we also find giant cells, either in the centre of the tubercle or arranged somewhat peripherally, while at the same time we notice that the death of tissue cells as well as of the cells that formed the centre of the tubercle has advanced, and that the tubercle is spreading peripherally in all directions, by the accumulation of small round cells in a zone about it. Frequently it can be made out that the various cellular elements of a tubercle, lie in a reticular net work. Some have claimed that this was due to post mortem changes, while others claim that

such a net work is produced, by prolongations of the giant cells. It is unessential.

At times there is quite an extensive formation of connective tissue about the tubercles, but rarely does this process become so well established that it is able to hem off the advance of the disease, except in very early cases where the lesions are few and the patients condition is of the best. Between tubercles there is seen an inflammatory condition of the tissue in which there is at times quite extensive formation of connective tissue.

The distinctive features of a tubercle by which you may define it and distinguish it from granulation tissue are these:- "The cells form a definite nodule which does not exceed a certain size, contains no new formed blood vessels, and in consequence at a certain stage of its development ceases to progress; that the nodule thereupon undergoes retrogressive changes, becomes necrotic, caseous and fatty, and furthermore that the nodule contains the specific organism of this disease, or the poisonous products of its development."

The other diseases which are classes as the granulomata are: Lupus, which is believed to be tuberculosis of the skin. Scrofula, so called, which we now consider as simply tuberculosis. Leprosy, Syphilis and a few others.

These diseases are all characterized by the presence of tissue closely resembling granulation tissue. They will not be considered separately, as the tubercle described will answer as a type for them all.

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The death rate from pulmonary tuberculosis is about one in seven, or a little more than 14%.

The cause of the disease we now know to be a micro-organism, described in 1882 by Koch, and called the tubercle bacillus

In the vast majority of cases this cause gains entrance to our bodies through the air we breathe.

It gets into the air by being spat up in large numbers by tuberculous persons. Drying does not kill this bacillus and in consequence it can exist for a considerable time in the dust of our streets, cars, etc., and be inhaled with the air. The bacilli are found clinging to small particles of dust and are inhaled with these. Knowing the cause of the disease, and how it becomes so wide spread we should endeavor to prevent its spread by adopting and teaching, proper methods of sputum destruction.

# THE INFILTRATIONS.

DEFINITION OF AN INFILTRATION:-"Any process by which a substance normally found in the body or foreign to it, is so to speak, poured into a tissue from without, which does not of necessity destroy its vitality, but frequently affects it mechanically by pressure or otherwise."-(Hamilton.)

We will consider only these two forms of infiltrations fatty, and amyloid.

## FATTY INFILTRATION.

DEFINITION:-"By fatty infiltration is meant the production of oil in the interior of a cell from materials from without."

In this form of fatty change the fat is as it were stored up in the cells of certain parts of the body. The change is one which is very frequently met with and exists as a normal process to a certain extent. After hearty meals for example there is more fat found in the cells of the liver than before meals. This, however is not pathological. It is only when the fat so stored up is

excessive in amount or pre-exists for a longer time than is proper, that the change passes into the domain of pathology.

That the process is not one of degeneration is proven by the fact that a careful study of the cells shows that though most of the space within their cell membrane is occupied by oil droplets, there is yet some protoplasm left in which one can see a nucleus which takes up all of the nuclear stains well.

The cells usually affected by this process are the connective tissue cells and the cells of many of the internal organs, as the liver, heart and kidney.

The chief causes of fatty infiltration are forced rest with a diet rich in carbonaceous material and such wasting diseases as tuberculosis. Alcohol also sometimes produces fatty infiltration when indulged in to excess, but it still more frequently produces that other form of fatty change, fatty degeneration.

If the cause of fatty infiltration be removed in time the cell affected entirely recovers, the fat being removed by a process of oxidation.

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## AMYLOID INFILTRATION.

This process has for some time been considered one of degeneration and has been so described. Now, however, it is looked upon as one of infiltration.

DEFINITION:- A condition in which there is deposited in the cells of a part, chiefly the connective tissue cells, and about them, a peculiar substance identical in composition with albumen.

The organs chiefly affected in this way are the spleen, liver, kidneys and intestines, though it is frequently found more widely distributed. When the condition is well marked it is recognizable with the naked eye, and the

organs so affected are larger and firmer and usually more elastic than normal.

The amyloid change is usually first noticed in the fibrous coats of the smaller blood vessels and about the walls of the capillaries, where by its increase it often seriously interferes with the blood supply of the part. At first this substance was supposed to be allied in composition to starch, but more recent investigation has proved that its composition is almost identical with that of albumen.

With certain chemical reagents it has characteristic reactions, for example, when treated with a solution of iodine and potassium iodide in water it takes on a beautiful mahogany color. Again when fresh tissues are treated with a solution of methyline green, or with a solution of gentian violet, two of the analine dyes, the amyloid material assumes a beautiful pink color, while the surrounding tissue is stained either a green, in the first case, or a violet color in the second. When either of these stains are used, which bring out the structure of the tissue well, it is seen that the amyloid material does not affect the parenchymatous cells of the part as was at one time supposed, but is confined, as just stated, to the connective tissue elements and only affects the other parts secondarily by pressure.

Amyloid changes are found either diffusely scattered through an organ or else confined to certain places. On the other hand a second form of amyloid change is sometimes met with which consists of firm deposits of considerable size, giving all the chemical reactions of amyloid material. These deposits are spoken of as "corpora amylacea". They are found in old hemorrhages, in syphilitic scars and diseased lymph-glands, and normally at times in the central nervous system.

As to the cause of this form of infiltration very little can be said. We know that it occurs in certain cachetic conditions of the system, notably in tuberculosis and

syphilis and it is most frequently associated with conditions of extensive suppuration of long duration. In some rare instances it has been noted to develope as rapidly as within two or three months.

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## CALCARIOUS INFILTRATION.

The process of salt deposit within the tissues has been spoken of for some time as calcarious degeneration, but it more nearly fulfills the conditions of our definition of an infiltration and will be so considered. The process is a purely passive one in which the cells of the tissues take no part. These tissues are, so to speak, petrified without loss of structure, by a deposit of the earthy salts from the blood. The salts so deposited are chiefly the phosphates and carbonates of lime and magnesia, and are probably held in solution in the blood, at least in part, by carbonic acid. In the majority of cases these deposits occur in tissues that are either dead or dying, in tissues in other words whose nutritive activity is below normal. In these cases it is probable that the diminished vitality and a retarded blood stream are instrumental in bringing about the deposit.

In other cases these calcarious deposits seem to occur because of an increase in the absolute amounts of these salts in the blood, a portion of the excess being precipitated more or less widely throughout the tissues. The deposits are most frequently noticed in the lymphatic glands and kidneys, and in the least active tissues of these organs, namely the connective tissue. Deposits of salts are also frequently found in the serous membranes of the joints.

In tissues which have previously undergone some fatty

degeneration and in the tissues of the aged these deposits of salts are very frequently to be found. For example, in the aorta the calcarious plates seen in advanced life are the result of salt deposit in areas of the aortic intima that have previously undergone fatty degeneration, the fatty patches being in many instances found side by side with the calcarious plates. The stiffened arteries also of the aged are the result of a salt deposit within the artery walls.

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## PIGMENTATION.

Pathological pigments found within the body may be classified as follows:

1. Pigments derived from the blood coloring matter
2. Pigments derived from extraneous sources.

This would not seem to cover the cases of bile pigmentation, but it should be remembered that the coloring matter of the bile is itself derived from the coloring matter of the blood.

Pigmentation by blood coloring matter:- Haemoglobin in solution.

The red staining of many tissues found at autopsy, such as the staining of the endocardium and the intima of the aorta is due to the absorption by these tissues of the haemoglobin dissolved out of the mass of red corpuscles in the heart and aorta. It should not be mistaken for congestion

Haematoidin Pigmentation:- This form of pigmentation is seen in old Hemorrhages. The haemoglobin of the blood undergoes chemical changes, resulting in the formation of crystals, or granules of a ruby red, or brick red color. The granules found in the liver cells in cases of passive congestion of some standing are haemotoidin granules.

Melanine Pigmentation:- This is a very common form of pigment and is found normally in the choroid, the rete Malpighii of the negroes skin and in other places.

Pathologically it is chiefly a degeneration of connective tissue growths, - sarcomata and fibrous tissues -. It is also found pigmenting the tissues in Addison's disease. It differs from haematoidin pigment in not being the product of extravaset blood but in requiring the action of living protoplasm for its elaboration. The cells of the tumors in which it is found (melanotic sarcomata) elaborate it independently of any blood extravasation. The pigment found in the spleen and other organs during malarial fevers is melanine pigment.

Bile Pigmentation:- The coloring from bile is yellow, or orange. The source of the color is a substance known as bilirubin which is probably of the same composition as haematoidin and is derived primarily from the blood.

Pigmentation by extraneous matters:- Chief among the pigmentations due to the entrance into the body of extraneous material is that pigmentation of the lung and bronchial glands from the inhalation of dusts of various kinds, chiefly coal dust.

Occasionally one meets also with cases of pigmentation from silver. This is shown generally by a blackening of the skin and is seen in cases where silver is administered internally for some time. Coloring from other metalic substances sometimes occurs.

# THE DEGENERATIONS.

DEFINITION:- "By a degeneration we mean any process whereby a cell-element or tissue undergoes such molecular changes that it can no longer maintain its functional activity, and either separates into its organic constituents or gives rise to the formation of a new product at the expense of its own substance". - Hamilton.

## FATTY DEGENERATION.

DEFINITION:- A condition in which the protoplasm of the cells affected becomes converted into oil, from which conversion the protoplasm is never restored.

This process differs essentially from that of fatty infiltration just studied in that the fat produced is not deposited within the cells, but is formed from the protoplasm of the cells, a true process of degeneration, the protoplasm being once lost is never reformed.

The causes of fatty degeneration are to be sought for in an altered condition of the blood, in some change in the constitution of the nourishment which is supplied to the cells. A deficient supply of oxygen plays probably the most important part in causing this change. To this diminished supply of oxygen we must ascribe both the disintegration of albumen and the formation of fat as well as the fact that the fat produced is not consumed, for it is by oxidation that the fat normally produced within the body is changed into other substances. From this point of view we would expect to find fatty degeneration occuring in those diseases in which the oxygen of the blood was lower than normal, and in all the various forms of anaemia whether it be one in which the number of oxygen carriers, the red corpuscles, is diminished, or

one in which the absolute amount of oxygen per corpuscle is lower than normal, we find these fatty changes. To this general cause of fatty degeneration must be added the excessive use of alcohol, and the action of certain poisons such as phosphorus and arsenic, as well as the action of prolonged high temperatures.

The results of fatty degeneration need hardly be mentioned. There is a permanent injury done to the part which brings its own results according to the part affected. For example fatty degeneration of the heart muscle may produce the most serious result, death, because of the great weakening which results. Fatty degeneration of other organs as the liver, may produce other more slowly developing results, which nevertheless are of a serious nature.

## ATROPHY.

DEFINITION:- "A diminution in size, or an absolute destruction of a part which results from direct and continuous over-pressure where the blood supply is not deficient." (Hamilton).

It will be seen that this definition does not include all cases of diminished size of a part from any cause whatever as we at one time held. For example the diminution in size which results from malnutrition would not come under the head of atrophy because here the diminution is due to a process of fatty degeneration which as we have seen is a distinct and separate morbid process. Atrophy then we are to consider as in reality a form of cellular degeneration resulting from continuous over-pressure and causing a diminution in the size or a total destruction of the part which the cells form.

When a cell becomes atrophic the first thing to be noticed is an irregularity in its outline and some

shrinking. The protoplasm then is seen to become granular and ultimately the cell is reduced to a small mass of granules, much smaller than its original self. These granules are ultimately absorbed and the cell in this way removed altogether. It is to be carefully noted that in this process there is no fatty degeneration whatever.

This definition of atrophy does not of course cover those cases of physiological diminution in size noted in certain organs such as the thymus gland in the child, and the uterus in the aged female. These are rightly not included as they are in no respect pathological and the term atrophy where applied to them is a misnomer.

The so-called atrophy of inaction, and the atrophy following the section of a trophic nerve, the so-called trophneurotic atrophy are in reality not atrophies in the true sense of the word as defined above, but are processes of fatty degeneration resulting in the diminution in the size of the parts affected because of a destruction of its cells.

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## CLOUDY SWELLING.

This is a term applied by Virchow to a condition of the parenchymatous cells of a part characterized by the formation of numerous small granules within the substance of their protoplasm giving them a hazy cloudy appearance. The process is one of cellular degeneration and by Virchow was considered as a manifestation of the cells of an inflammatory process. The condition is sometimes spoken of as parenchymatous degeneration.

Whatever may be the cause, if it ceases to act before the cells are too much injured, they will return to their normal healthy condition, but if the cause is not removed the cells ultimately undergo fatty degeneration, and break up into small masses of fatty granules.

# NECROSIS.

Death of the organism as a whole is spoken of as somatic death. Besides this death as a whole we may have death of individual organs, or of portions of organs and tissue. This local death is spoken of as necrosis. The causes of necrosis are in brief:— 1. Arrest of the circulation. 2. Mechanical and Chemical injuries. 3. Thermal injuries.

All causes producing arrest of the circulation produce necrosis in the tissue from which the blood is cut off. Some tissues suffer more quickly than others from this lack of nutrition. Brain tissue suffers very quickly, while muscle tissue can withstand the loss of nutrition for some time. A typical example of necrosis from cessation of the circulation is seen in infarcts.

Mechanical, chemical and thermal injuries it is easily seen can produce local death of tissue.

The constant result of local death is a more or less severe inflammation in the healthy tissue surrounding the necrotic patch. Owing to this the necrotic tissue is more or less completely hemmed off from the surrounding parts, and not infrequently is it cast off from the plant by this process.

The chief form of necrosis which we meet with is known as coagulation necrosis. This occurs in two ways. First, in certain fluids of the body, as the blood, lymph, and the fluids in certain inflammatory exudations, there occurs a fibrinous, granular or homogeneous coagulation, accompanied by the death of cells. Secondly, cells and intercellular substance as they die become firm and solid and coalesce into a homogeneous mass. In the first instance the material that was formed in the fluids was fibrin. In the second, the substance into which the cells was converted was also fibrin. In this case we have the

dying cells of the part giving up fibrin ferment, and at the same time they are soaked with the lymph, rich in fibrinogen, the result being coagulation. In a tissue that is undergoing coagulation necrosis the cells are seen to maintain their form and general appearance, but to be more granular than normal. They stain poorly and after a time their nuclei disappear entirely. A mass of such necrotic tissue may after a time undergo other changes, becoming fatty, liquified, etc.

## CASEATION.

This occurs in two forms, one of which is a process of coagulation necrosis, and the other of fatty degeneration. In the form due to coagulation necrosis the mass is firm, yellowish white, and resembles new dairy cheese. The tissue may assume this cheesy appearance in one of three ways:- 1. It may gradually, as a whole assume a firm whitish homogeneous appearance, losing its nuclei and staining more and more poorly. 2. Detached portions of the necrotic mass may take on this appearance and ultimately fuse into one homogeneous mass. 3. The necrotic mass may be first dissolved and from the solution fibrin be deposited which then fuses into the cheesy mass.

The second form of caseation, is softer and more nearly resembles cream cheese. It is due either to the fatty degeneration of tissue or of exudates. The tissue or the exudate gradually breaks up into a mass of fatty granules and detritus, and the water is slowly absorbed, the mass becoming more and more inspissated and finally dull, opaque in appearance.

## SOFTENING.

This may either proceed or follow coagulation. In the case of the coagulation of the blood there is first a softening and breaking up of the cells and then coagulation, while in the case of a thrombus which has formed, there is before the softening of the thrombus, the coagulation. In the case of a blister raised by heat, there is first softening and liquefaction of certain cells before there is any coagulation.

## GANGRENE.

Is divided into moist and dry variety. The dry gangrene, sometimes spoken of as mummification. Here there is first necrosis of tissue in parts which are exposed to the air, followed by rapid evaporation of liquids and the drying of the part.

Moist gangrene is necrosis followed by putrifaction or decomposition. It is due to septic organism having gained entrance to the necrotic tissue and by their action producing solution, liquefaction and the complete destruction of the part.

# CELL DIVISION

All hypertrophy, hyperplasia and new growth are due to an increase in the number of cells which compose a part. This increase is only brought about by a repeated subdivision of the cells which compose the tissue.

Cell division can take place in one of two ways. The first and most simple process is a mere splitting of the cell and its nucleus into two parts each becoming a separate cell. This process is spoken of as "direct cell division" and is not so common as the second method which is known as that of "indirect cell division" or Karyokinesis.

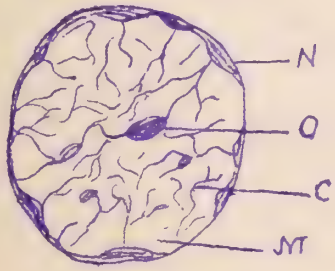



Fig. 9.- Resting Nucleus.

N. Nuclear Membrane
O. Nucleoli
C Nuclear Net Work (Chromoplasm
M. " Matrix

In order to understand this process one must know the structure of the resting nucleus. This in brief is as follows. The nucleus is bounded by a definite wall which encloses the nuclear contents which are of two kinds, formed and amorphous. The former is spoken of as the chromoplasm and the latter as the nuclear matrix. The chromoplasm derives its name because of its affinity for certain coloring matters, and it usually takes the form of a net work of fibers communicating externally with the nuclear wall. One or more strongly refractive granules are usually found suspended in the nuclear matrix either isolated or connected with one another by the fibers of the chromoplasm. These are the nucleoli.

## KARYOKINESIS.

The first step in the process of indirect cell division is the drawing in of the main chromatic fibers and of

Fig 10. Karyokinesis
Nucleus viewed from the pole. Chromatic filaments forming about pole

their fine branches. These thereby become somewhat thickened and after a short time are seen to assume the shape of loops or "hair pins" and ultimately to become arranged about one pole of the nucleus with their looped ends pointing toward the pole and their free ends interdigitating at the opposite pole.

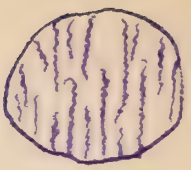


Fig. 11. Karyokinesis
Nucleus viewed from the Anta-pole. Interdigitating ends of Chromatic loops.

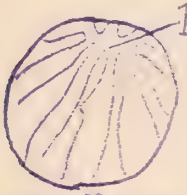



Fig. 12 Karyokinesis
Nucleus with ... pole

At this time there appears at the pole within the looped ends are forming a peculiar body spoken of as "spindle shaped body." At first it occupies a more or less horizontal position. But slowly becomes more and more upright, while at the same time the looped ends of the Chromatic filaments move downwards and their free ends upward and outward, so that they come to lie about the centre of the spindle and are at right angles to it.

Fig. 13. Karyokinesis
Spindle appearing at pole "Monaster Stage"

Fig: 14 Karyokinesis
Spindle upright Chromatic loops arranged about middle split longitudinally

Fig 15. Karyokinesis
Beginning separation of Chromatic loops

Now these Chromatic filaments cleave lengthwise and soon after the upper half travels upward toward the pole they left and the lower half travels downward to the opposite pole. For a time their free ends intermingle about the equator of the spindle, but soon part and are drawn toward the two poles. Here the two sets of loops are arranged as was the original set from which they were derived and we have what is spoken of as the

"dyaster stage" or the two star stage. These two sets of loops soon put out branches, become convoluted and skein like and each resembles a resting nucleus.

Now the protoplasm of the cell divides and each half receives one of the newly formed nuclei and we have two cells derived from one

Fig. 16. Karyokinesis
Further Liberation of Chromatic Loops.

Fig. 18. Karyokinesis
Cell in "Dyaster Stage"
Faint remains of Spindle

Fig. 17. Karyokinesis
Spindle with Chromatic loops arranged about each pole.

Fig. 19. Karyokinesis.
Cell with two young Nuclei about to divide.

# HYPERTROPHY.

Hypertrophy may be defined as that condition of increased size of a part due either to an increase simply in the size of its constituent cellular elements, or to an increase in their size and in their number, but hypertrophy does not include those cases of increased size due solely to an increase in the number of cells within a part. This latter process we term hyperplasia.

Most of the cases of hypertrophy met with, as for example the hypertrophy of the biceps, or of the heart from over-work are examples of increase in size due to both an increase in size and in number of the cellular elements of the parts affected.

The chief causes of hypertrophy may be mentioned as over-work, increased supply of nourishment, and intermittent pressure. A familiar example of the latter is seen in the ordinary corn.

The results of hypertrophy, are if anything, increased functional activity of the part.

# TUMORS.

A more proper term to use than tumor would be neoplasm for the word tumor signifies merely a swelling and is usually applied to localized projections. A neoplasm generally forms a tumor, though not always, and a tumor is not always due to a neoplasm. For example an abcess may form a tumor, but it is not a neoplasm. In using the word tumor then, as it is in common use, we will be understood to mean neoplasm.

DEFINITION:- A neoplasm, or new formation is "the excessive production of some tissue naturally existing in the body during foetal or adult life, either locally where such tissue is normally present, or in some part of which it is not an actual constituent." - (Hamilton).

## CLASSIFICATION OF TUMORS.

Tumors are classified according to the character of the cells which compose them, the character of the cells depending upon their origin from one or the other of the embryonic germ layers. One large class of tumors is composed of cells derived from the middle germ layer, or MESOBLAST, and hence are spoken of as mesoblastic tumors. Another large group of tumors are composed of cells derived chiefly from the uper germ layer, the EPIBLAST, and are spoken of as epiblastic tumors.

The mesoblastic germ layer gives rise in the adult to such tissues as fibrous tissue, bone, cartilage, muscle, blood, and lymph vessels, etc. We find tumors also which are composed of these same tissues and they are therefore classified as mesoblastic tumors. Such tumors are the fibromata, composed of fibrous tissue; the osteomata, composed of bone; the chondromata, composed of cartilage;

Such mesoblastic tumors as these composed of simple tissues exactly like tissues found already formed in the adult body are spoken of as "simple tissue tumors" or as "histoid tumors. They are all benign, that is harmless except for the effects which may be produced by the pressure they exert.

There are however other mesoblastic tumors which, though composed of cells derived from the middle germ layer, do not resemble in structure any formed tissues. These are known as the sarcomata, and may be defined as "tumors composed of an embryonic connective tissue which shows no inclination to fulfill its ultimate developmental intention" (Hamilton). These tumors are malignant.

The second great class of tumors the epiblastic tumors embraces the various kinds of cancers, or carcinomata, cutaneous horns and warts; papillary epithelial growths, the papillomata; certain forms of glandular tumors, the adenomata; tumors of nerve tissue, the neuromata, etc. Of these the carcinomata, and adenomata are malignant, and the others as a rule benign.

## SIMPLE TISSUE, OR HISTOID TUMORS.

These, as it has been said are the tumors which in their structure resemble some formed tissue derived from the mesoblastic germ layer. They are all benign growths and form well defined circumscribed tumor masses. The chief forms of these tumors are as follows:

### FIBROMA - FATTY TUMOR.

Definition:- A tumor composed of white fibrous tissue.

Sites:- These tumors may grow wherever fibrous tissue exists. They are most commonly found in the skin, sheaths of the tendons, fasciae, and muscles mammary.

Microscopically:- They are made up of bundles of white fibrous tissue, interlacing in all directions. Along these bundles can be seen spindle shaped nuclei. Where the tumor is rapidly growing one may see large numbers of young, round & oval cells. If such a tumor be removed but partially & it is young and growing it will return. It is not however of malignant growth.

## LIPOMA - FATTY TUMOR.

Definition:- A localized hypertrophy of fat tissue.

Sites:- Most common in the sub-cutaneous fat, especially in those parts which are pressed upon. They are rarer in internal parts, but may occur wherever there is fatty tissue.

Microscopically:- Simply lobulated masses of fat tissue usually sharply defined and often encapsulated. Non-malignant.

## CHONDROMA - CARTILAGINOUS TUMOR.

Definition:- Masses of new tissue composed either of hyaline, or white fibro-cartilages.

Sites:- The hyaline variety is usually met with growing from bone, not from cartilage and is generally found growing from bony prominences. The fibro-cartilage variety occur more particularly in the capsules of joints and in ligaments.

Microscopically:- The hyaline variety is composed of islands of hyaline cartilage bound together by fibrous septa in which run the blood vessels that nourish the tumor. In the fibro-cartilage variety the tumor is not subdivided into lobules, but is a uniform mass of white fibro-cartilage.

These tumors are non-malignant though sarcoma-round may occur if not completely removed.

## OSTEOMA – BONE TUMOR.

Definition:- "A tumor mass of bone, developed by a process of growth without the occurrence of inflammation". (Hamilton.)

Sites:- 1st. From the vault of the skull, or wall of the external auditory meatus. This variety is very hard and dense. (osteoma durum.) 2nd. From the epiphyses of the long bones. The tumor originates in cartilage and ultimately becomes assified. The bone presents a cancellous structure like spongy bone. (osteoma spongiosum). 3rd. There is a variety very similar to the above in which the cancellous spaces are filled with marrow. (osteoma myeloides).

Osteoma may at times develope from fascia and tendons. Osteoma are non-malignant growths.

## MYOMA -- MUSCLE TUMOR.

Definition:- Tumors composed for the most part of unstriated muscle fibers, though occasionally there is a congenital variety met with in which the fibers are striated.

Sites:- Unstriated; in parts where muscle tissue normally occurs, and at times in the prostrate of old people. Striated; chiefly in connection with the Kidney and testis, and is always congenital.

Microscopically:- Unstriated; masses of unstriated muscle fibers. Striated; masses of spindle cells, most of which show the transverse striations. Sometimes spoken of as myo-sarcomata.

## BLOOD-ANGEIOMA.

Definition:- A tumor composed of dilated blood vessels.

Varieties:- Simple angiomata, or varicose dilitations of veins or capillaries. Cavernous angiomata, tumors composed of masses of new formed blood vessels.

Sites:- The simple variety occurs most frequently on the

skin of the face. They are the so called naevi. The second variety, the cavernous angiomata occur chiefly in the liver.

Microscopically:- The simple variety consists of greatly dilated and varicose veins and capillaries and are congenital. The cavernous variety in structure closely resemble corpus cavernosum of the penis and constitute at times large masses embedded in the substance of the liver.

## LYMPHANGEIOMA.

Definition:- A tumor composed of dilated lymph vessels.

Sites:- Chiefly in the subcutaneous, and submucous tissues.

Microscopically:- They consist of a series of branching dilated varicose lymph channels. Little is known of the cause of this condition.

## THE SARCOMATA.

The chief varieties of sarcomata, a definition of which has already been given, may be briefly mentioned as follows.

SMALL ROUND CELL SARCOMA:- A tumor composed of small embryonic cells, of meloblastic origin, and of about the size of white blood corpuscles. They are chiefly found in the subcutaneous tissue, attached to bone, or springing from the neuroglia of the brain or cord. It is usually soft in consistency. It has sharply defined borders and can be completely removed. Like all sarcomata it is malignant.

LARGE ROUND CELL SARCOMA:- In this form of tumor the cells are a number of times larger than those

in the foregoing variety. They are comparatively rare.— Maligniant.

THE SPINDLE CELL SARCOMA:— They are probably the most common of the sarcomatous tumors. They are usually found in dense fibrous tissue structures. In structure they are composed of masses of spindle cells. Three varieties are recognized. (1) The small spindle cell sarcoma, which is the most common. (2) The large spindle cell sarcoma, and (3) The oat-seed-like spindle cell sarcoma which is made up of masses of small spindle cells of about the shape of oat-seeds. The spindle cell sarcomas are the hardest in consistence of all this class of neoplasms and are the least maligniant. At times they closely resemble the simple fibroid tumors.

GIANT CELL SARCOMA:— These tumors are sometimes spoken of as myeloid sarcomata. They are most frequently found growing from bone or periosteum. As a rule they are soft tumors and when growing from bone they tend to grow into the bone and in this way frequently lead to fracture of the same. The tumor in the main is composed of spindle shaped cells in which there are found a large number of multinucleated giant cells. This neoplasm while at times appearing to be maligniant is not markedly so. The chief damage done is local. Rarely if ever is the tumor composed entirely of giant cells.

Other forms of Sarcomata are met with, such as the Angeioma-Sarcoma, Alveolar-Sarcoma, etc. which will not be described.

## EPI- AND HYPOBLASTIC TUMORS.

Aside from the cutaneous horns and warts that are so frequently met with the most important forms of epi- and hypoblastic tumors, those formed from the epiblast, or from the hypoblast, the upper and under germ layers, are cancers, or carcinomata and the adenomata, or tumors composed of formed glandular tissue.

# CANCER OR CARCINOMA.

DEFINITION:- "A tumor formed of any tissue whose fibrous interspaces and lymphatic vessels are infiltrated with actively proliferating epithelial cells". (Hamilton)

The term epithelioma is to be preferred for all cancerous tumors as the meaning of the term is in harmony with the structure and origin of these growths and is a better term than cancer, the original meaning of which is a crab.

SITES:- These tumors may occur in almost any part of the body, but are much more commonly met with in certain localities, such as the mammae, the pyloric end of the stomach, the uterus, etc.

STRUCTURE IN GENERAL:- According to the above definition all these tumors are composed of tissue in the fibrous meshes of which and in whose lymphatics one finds large numbers of rapidly reproducing epithelial cells. In many of these cancerous growths these epithelial cells are arranged between the meshes of fibrous tissue in such a way as to indicate the beginning formation of a glandular like structure. The process however is never carried to completion, but the crude formation is repeated indefinitely. All Cancers tend to infiltrate surrounding parts, have as a rule poorly defined borders, tend to ulcerate and are frequently hard. The metastatic cancers which form are identical in structure with the primary growth. The relative proportions of stroma and epithelial cells determine the consistency of these growths.

# ADENOMATA.

These tumors are essentially cancers, that is they are epithelial growths which correspond to the definition given for cancers, but instead of having the unformed glandular structure so common with ordinary cancerous

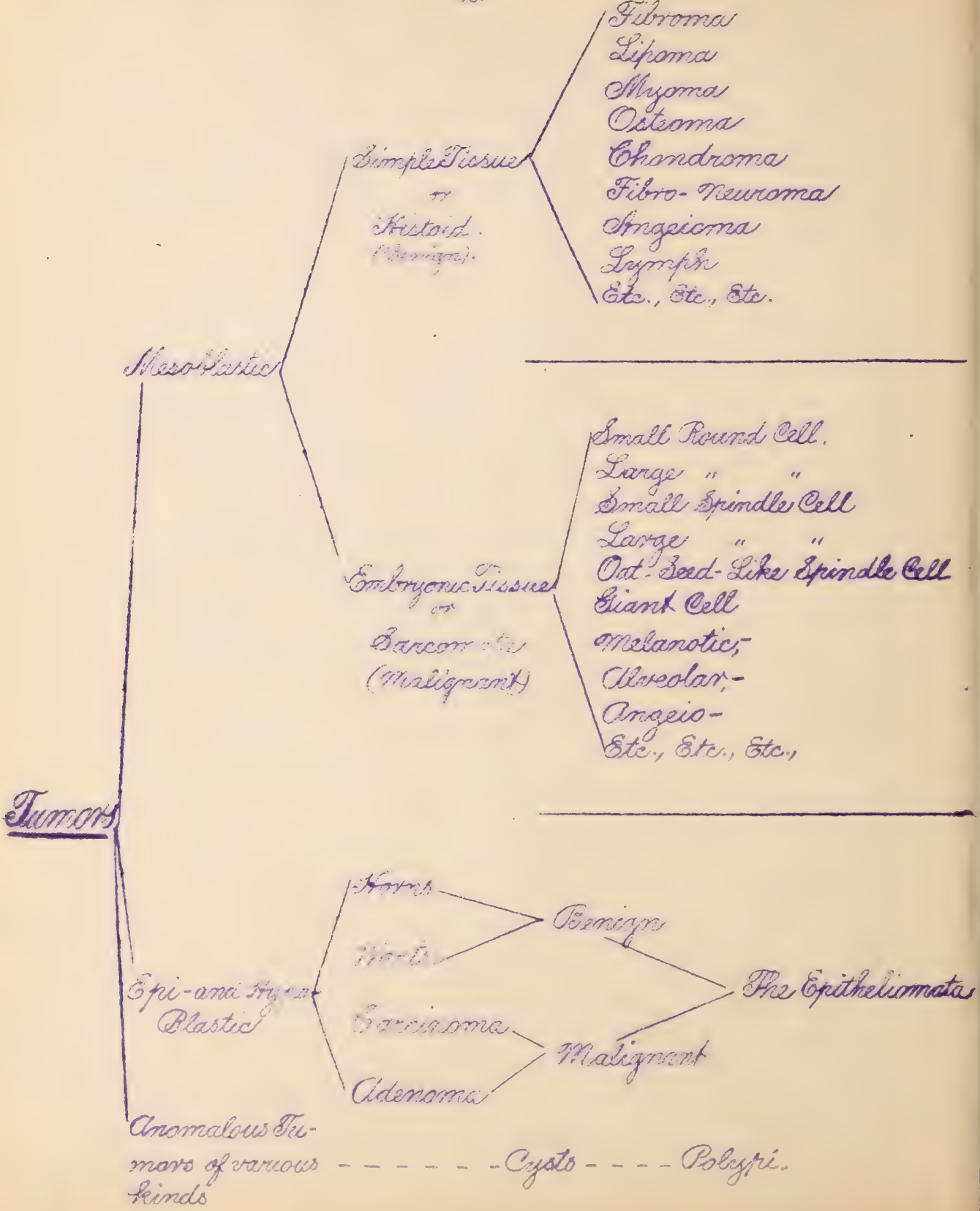
growths, the epithelial cells are all arranged as in fully formed glandular tissue. There is a repeated formation of glandular acini and the entire tumor mass is composed of these masses of glands. They are extremely malignant and are most commonly found in the walls of the stomach and intestines.

## MALIGNANT AND BENIGN TUMORS.

Malignant tumors are harmful, while the benign tumors if they produce any injury whatever, injure only the part in which they grow, because of the pressure which they exert and the interference with the function of the part which their presence causes.

The essential characteristics of malignant tumors are 1. the power to form metastatic growths, that is tumor masses identical in structure with the original tumor, but situated at some distance from it. 2. The tendency to return after apparently complete removal; and, (3). a tendency to infiltrate surrounding parts.

What the exact nature of a tumor is which determines its malignancy is as yet disputed. It is however something given off from the tumor and from its metastatic growths, which can be conveyed in the lymph and blood currents. Some consider that it is the peculiar property of these malignant tumor cells to rapidly multiply when planted, so to speak, in tissue soils, and that is through the escape of these cells into the blood and lymph currents and their lodgement elsewhere that metastatic growths occur. Others of late are attempting to prove the parasitic origin of certain tumors. It is probable that in the great majority of cases the first view is the correct one.


Tumors
Meso-Plastic
Simple Tissue
or
Histoid.
(Benign).
Fibroma
Lipoma
Myoma
Osteoma
Chondroma
Fibro-Neuroma
Angeioma
Lymph
Etc., Etc., Etc.
Embryonic Tissue
or
Sarcoma
(Malignant)
Small Round Cell.
Large " "
Small Spindle Cell
Large " "
Oat-Seed-Like Spindle Cell
Giant Cell
Melanotic,-
Alveolar,-
Angeio-
Etc., Etc., Etc.,
Epi-and Hypo-Blastic
Horns
Wart
Carcinoma
Adenoma
Benign
Malignant
The Epithelimata
Anomalous Tumors of various kinds
Cysto
Polypi.

A

LABORATORY MANUAL

— of —

# Pathological Anatomy

SECOND EDITION

FIRST PART.

DISEASES OF

# The LIVER, SPLEEN & PANCREAS

1895

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# DISEASES OF THE LIVER.

## Normal Structure In Brief.

*For a full account of the anatomy and histology of the liver read either Quain's or Gray's Anatomy.*

The liver is the largest organ in the body. Its average weight in the adult is about 3 pounds (1,400 grams). It lies chiefly on the right side of the abdomen, though a portion of the lesser, or left lobe passes the median line and lies upon the left side.

The organ is covered by a thin glistening fibrous tissue capsule composed of a superficial and of a deep layer. The superficial layer is continuous with the peritoneum, while the deep layer dips into the organ in innumerable places and forms its supporting structure. Where ever these small bands of fibrous tissue from the deep layer of the capsule enter the liver they are accompanied by branches of the portal vein, the hepatic artery, and the bile ducts, the whole group being known as a portal system or a portal canal.

FIGURE 1.
A portal canal = P.V. Portal Vein. HA. = Hepatic Artery. BD. = Bile Ducts

The great mass of the liver is made up of liver cells. These are finely granular, polygonal, nucleated bodies, arranged in a lobular manner with reference to the terminal branches of the hepatic vein. In many animals this lobular arrangement is very distinct, small bands of connective tissue separating one from another, but in man no such definite demarcation can be seen. Each lobule consists of a mass of liver cells, arranged roughly in columns about a central vessel, the intralobular hepatic vein, into which open many minute capillaries bringing the blood

from the portal vein and the hepatic artery. While the terminal branches of the hepatic vein are found in the centre of the lobules, about their periphery, at the points of contact of several lobules will be found the portal systems.

Circulation:— The circulation within the liver is of importance, the organ having two distinct blood supplies, one arterial and the other venous.

Arterial blood is supplied by means of the hepatic artery, the small branches of which follow, as already stated, the ramifications of Glisson's capsule.

Venous blood is supplied by means of the portal veins which also ramify with the hepatic artery, and bile ducts in the portal systems. Blood from these two sets of vessels finds its way by a series of intralobular capillaries, which lie between the cells of the outer portions of the lobule, into the capillaries already mentioned as lying between the cells of the inner portions, and which empty their blood into the intralobular hepatic veins, or the central veins as they are often called. From these veins it is taken up by larger vessels lying beneath the lobules and hence called the sub lobular veins, which ultimately empty as the hepatic veins into the inferior vena cava.

Origin of the Bile Ducts:— The bile ducts originate within the liver lobules. Where two or more liver cells are grouped together the minute radicles of these ducts begin. At first these small channels are strictly intercellular, each liver cell being grooved along its sides, and where two cells come into apposition a channel is thus formed. Innumerable channels

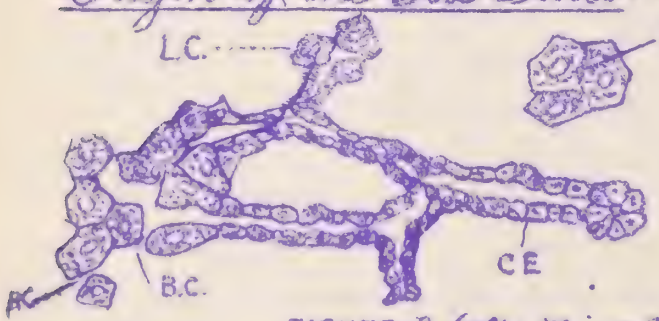



FIGURE 2. (after Klein & Smith)
Commencement of bile-ducts.
B.C.- Bile Canaliculi between liver cells.
L.C.- Liver Cells slightly modified just before beginning of bile duct.
C.E.- Cubical Epithelium of a large duct.

so formed communicate in all directions forming a most extensive net-work of capillary vessels. As these channels increase in size they cease to be intercelular as at first and become lined by a layer of small flat cells which as the duct increases in size increase also, until ultimately the bile ducts are well defined tubes lined by a cuboidal epithelium seated upon a basement membrane about which there is developed a slight muscular coat. According to some observers the bile capillaries begin as minute channels within the protoplasm of the cells and then come to lie as just mentioned in grooves between several cells.

Until recently the portal canals were looked upon as marking the periphery of lobules, while the hepatic veins indicated their centre. According to some observers however this is considered incorrect. These authors look upon the hepatic veins as marking the periphery, while the portal canals indicate the centre of the true hepatic lobules. About the hepatic veins they claim a series of glandular acini are arranged which converge with those from other hepatic veins, to a portal canal as a centre. Each acinus is conceived as composed of a mass of liver cells grouped about a bile duct into which empties an extensive capillary system. The liver cells secrete the bile which is taken up by the minute capillaries and conducted to the main bile duct, the duct for the gland and which lies in a portal system with its portal vein and hepatic artery.

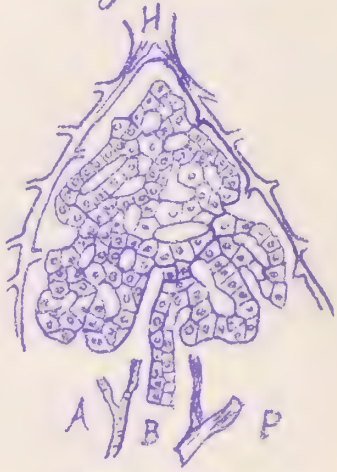



FIGURE 3.
Sabourins Scheme of a Hepatic Acinus.
A-Hepatic artery. B. Bile duct.
P-Portal vein. H-Hepatic vein.

Pathological Zones:- Returning however to the older conception of the liver lobule, we may for convenience sake divide it into three zones, each occupying about one-third of its area. Of these the outer is spoken of as the peripheral, or portal vein zone, and in it we first find the fatty changes to which the liver is subject.

The middle third of the lobule is spoken of as the intermediate zone, or the zone of the hepatic artery, because here the portal vein capillaries and those from the hepatics are are supposed to anastomose. In this area we first find waxy changes. The inner third is spoken of as the zone of the hepatic vein, or central zone. In it are found the capillaries which empty into the central vein and here we meet the first evidences of passive congestion of the liver.

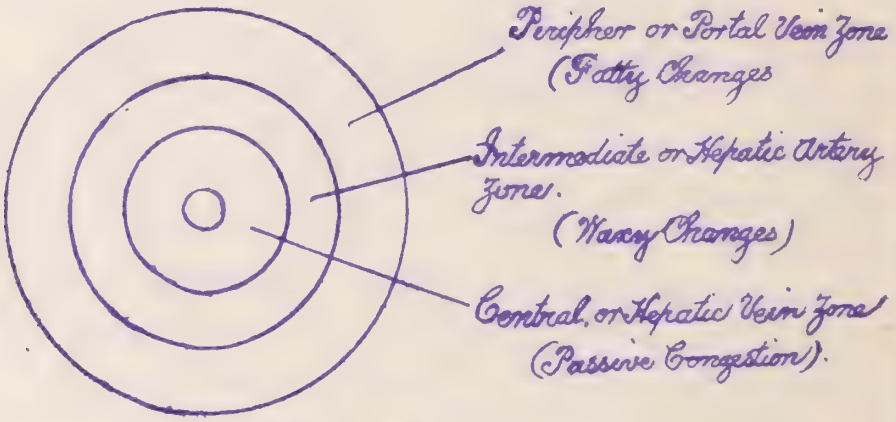



FIG. 4. Diagram of Lobular Zones.

# CLOUDY SWELLING

Synonyms:- Parenchymatous inflamation. Parenchymatous Degeneration.

Definition:- A change in the liver characterized by a swollen granular condition of the liver cells.

Etiology:- Cloudy swelling has been described by Virchow as a parenchymatous inflamation of the cells of the part, an early expression on their part of an inflamatory process. (See Lecture notes on Cloudy Swelling). The condition is found accompanying septic fevers and all acute febrile affections.

Pathological Anatomy:- Very little can be said of the gross appearances of a liver which is in a state of cloudy swelling. In consistency the organ is rather flabby and the color is somewhat lighter than normal. The capsule is usually rather tense, indicating that the liver is somewhat enlarged.

Pathological Histology:- Under the low power of the microscope it can be seen that the liver cells apparently take up more space than usual and that in consequence the capillaries are compressed and contain but little blood. This fact probably accounts for the somewhat lighter color of the organ. Some proliferation of the connective tissue may be seen but it is not marked.

Under a higher magnification the cells, especially those of the portal zone, (the outer zone) appear somewhat enlarged; they are more rounded than normal and within each can be seen numerous small granules which give to the protoplasm a cloudy look. The nucleus too is somewhat indistinct. If the condition be advanced many of the cells will be found broken up into a fatty granular detritus. Fatty degeneration is one of the terminations of cloudy swelling though if the cause be removed before the process is too far advanced the cells will return to their normal condition.

# Chronic Passive Congestion

Synonyms - Venous Congestion. Nutmeg Liver. Central Red Atrophy (Virchow) Cyanotic Atrophy.

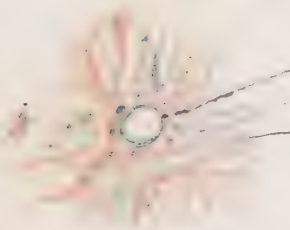
Definition: - A condition of the liver characterised by an over-fullness of the hepatic venous system with blood, and in advanced cases by atrophy of the liver cells from pressure.

Etiology: - Chronic passive congestion is a condition brought about purely by mechanical means. Anything which will hinder the efflux of blood from the hepatic veins will produce it. Thus for example, the most frequent cause is disease of the mitral, or of the tricuspid valve allowing at each systole the back flow of a certain amount of blood. If the lesion be tricuspid regurgitation the liver soon becomes affected, for at each cardiac contraction a portion of the blood which should have gone on to the lungs is forced backward into the right auricle thereby preventing the entrance of a certain amount of venous blood from the venae cavae. This produces a condition of over-fullness of the inferior vena cava, which in turn reacts upon the hepatic veins which opens into it. (Explain the way in which mitral regurgitation produces Chronic passive congestion of the liver.)

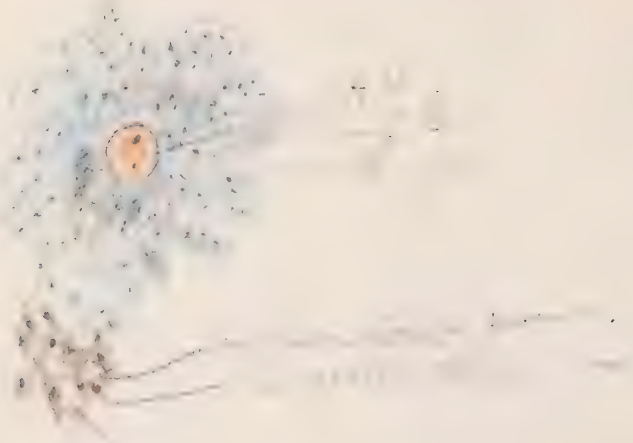
Of other conditions which can cause venous congestion of the liver, lung changes which interfere with the pulmonary circulation, are the most common. Thus chronic interstitial changes and emphysema by causing a hindrance to the passage of blood through the lungs force a certain amount back upon the right heart and upon the venae cavae. Tumors pressing upon the inferior vena cava will also produce congestion of the liver.

Pathological Anatomy: - We may recognise at least two

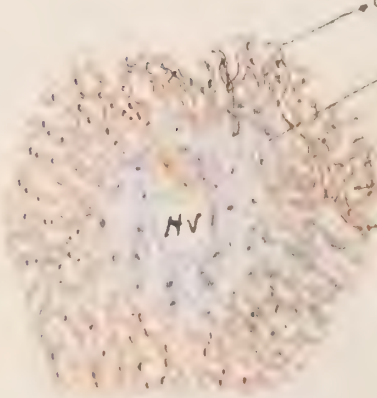
Portion of Lobule, Under Low Power.




Portal System, under Low Power



Lobules showing congested & non-



stages in this condition, an early stage and an advanced. In the early stage we find the liver somewhat enlarged, its capsule tense and glistening and of a purplish red color. Its consistency is now not much altered. In the advanced stage, we find that the organ has shrunken to something less than its normal size, its capsule is no longer glistening, but is wrinkled and dull. In consistency it is considerably firmer than normal.

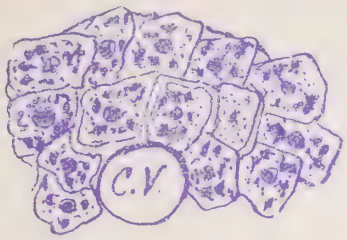



FIGURE 5.

Early "Nutmeg" liver, unstained.

Portion of lobule showing pigment in the cells around the central vein.

Upon section, in both stages, there is a plentiful escape of blood and upon closely examining the cut surface a distinct mottled appearance is made out. The centre of each individual lobule is seen to be of a deep red color while surrounding this can be seen a band of lighter tissue. If in the early stage, the central red portion will be small and the surrounding band of lighter tissue broad; while in the advanced stage the central portion will be large and the band of light tissue narrow. This mottled appearance has lead to the name "Nutmeg Liver" for this condition.

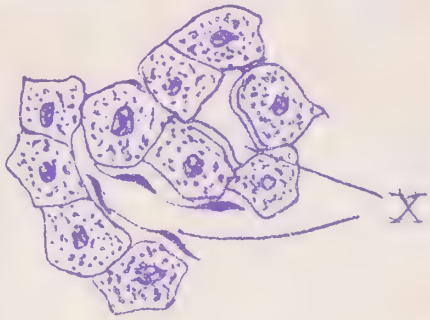



FIGURE 6

× Chronic Passive Congestion.

X – Dilated Capillaries showing the cells of their walls at some distance from the liver cells (×600)

Pathological Histology: In very mild grades of this disease where there may be very few grass appearances, or even none at all to call attention to it, we find upon microscopic examination a slight dilatation and engorgement of the capillaries in the zone of the hepatic vein, the centre of the lobule with a deposit of golden-brown pigment in the liver cells about them

As the disease advances the capillaries become more and more engorged and distended, and the columns of liver cells narrower. Already examining the capillaries one can make out their walls with much greater ease than in the normal organ, and in many places a distinct space between them and the columns of liver cells can be demonstrated.

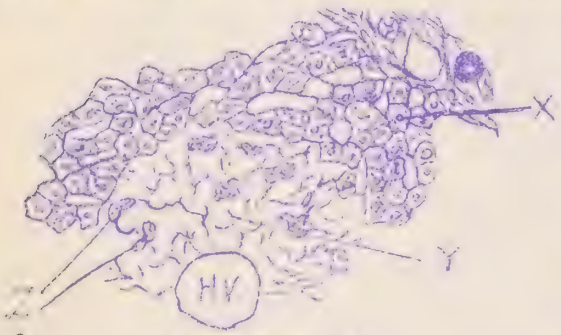



FIGURE 7.

Portion of a lobule in Advanced Central Red Atrophy

H.V.- Dilated Central vein. X.- Zone of comparatively normal liver cells at periphery of lobule. Y.- Atrophic Granular liver Cells. Z.- Central portion of the lobule in which all liver cells have been destroyed showing a reticulum of Capillary Walls. The blood has been washed out in preparation.

In the most advanced stages we find in the zone of the hepatic vein, a tremendous dilatation and engorgement of all the capillaries, and either an entire absence of liver cells, or the shrunken pigmented remains of a very few. The centre of each lobule seems to be converted into a mesh-work of blood containing tissue. Here and there where the blood has escaped from the capillaries and their walls have collapsed, an appearance very much resembling connective tissue is produced. In some instances where we would expect to find only a mass of blood, we find that, due probably to some fault in the fixing, or to the process of hardening and embedding, all the blood has escaped, and we only see a central reticular mesh-work of empty capillaries. As we proceed outward from the central area we come upon liver cells in all stages of degeneration and atrophy from the pressure exerted upon them by the distended capillary vessels, until finally at the extreme periphery, there is a narrow band of fairly normal cells.

# Fatty Infiltration of the Liver.

Synonyms:- Fatty Liver. Steatosis of the Liver.

Definition:- A condition of the liver characterized by a deposit of fat within the liver cells.

Etiology:- Accumulation of fat within the cells of the liver is in part a physiological process. Normally after a hearty meal there is an increase of fat within the liver which disappears within a short time. When however the amount of such fat increases beyond a certain point or remains unconsumed beyond a certain length of time the process passes beyond the physiological limit and becomes pathological.

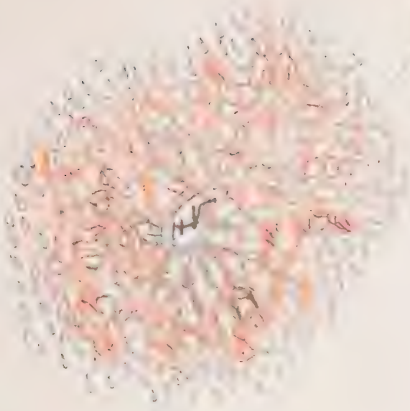
Fatty infiltration is met with under a variety of conditions. It is very frequently found in corpulent persons. All conditions which diminish oxidation tend to increase the deposit of fat in the liver as elsewhere, and such food with lack of exercise combined therefore favor its formation. In these cases it indicates a condition of the body in which there is a surplus of carbonaceous nourishment over what is required by the system.

Alcohol, also when used to excess leads to fatty infiltration of of the liver. This is probably to be explained by the fact that alcohol is more easily oxidized than the hydrocarbons of the body and hence is used up in preference to them while the excess of unconsumed carbonaceous matter in the body accumulates and is stored up chiefly in the liver and adipose tissue.

The most pronounced cases of fatty infiltration are found in the livers of those who suffer with chronic pulmonary tuberculosis. The probable explanation of this is that the diseased lung is insufficient for thorough oxidation within the system and in consequence the fat accumulates. Why the liver should be chosen for the storehouse for fat and not other portions of the body is not as yet understood.

PATHOLOGICAL ANATOMY.- The liver which is infiltrated with fat is uniformly increased in size. Sometimes this increase is enormous, the liver being several times its normal size. Although so large and in consequence as a whole much heavier than normal, pieces of tissue, cut away, and sometimes the entire organ, will float in water. Usually the border of the liver is smooth and sharp, though occasionally it is swollen and rounded. The capsule, on account of the increased size is tense and glistening. In consistency, the organ is quite flabby. It usually pits on pressure the indentation remaining for sometime after the pressure is removed. On section the tissue is of a decided mottled yellow color. In the early stages the yellow portions can, with the naked eye, be seen to form rings about the periphery of the lobules, the central portions being normal in color. In more advanced cases the entire surface presents the same yellowish hue. The blood vessels usually contain less blood than normal and the tissue is quite friable.

PATHOLOGICAL HISTOLOGY Under the microscope it can be seen that in the earlier stages the deposit of fat is confined to the periphery of the lobules. In rare cases there is also found a similar deposit within the cells about the central vein, the intermediate tissue being normal. Under the low power the droplets of fat will be found to vary considerably in size, the largest occupying the cells at the extreme periphery while the smaller ones are found farther within the lobule. Each drop appears as a bright spot, or clear space and upon focusing upon it one can see either a bright center with a dark border, or by changing the focus just the opposite. Under the high power, the liver cells which are loaded with fat will be found to have lost their polygonal shape somewhat. In some there will be found considerable granular looking protoplasm and only a few small droplets of oil, while in



others the several small drops seem to have flowed together into one large drop which now occupies nearly the entire

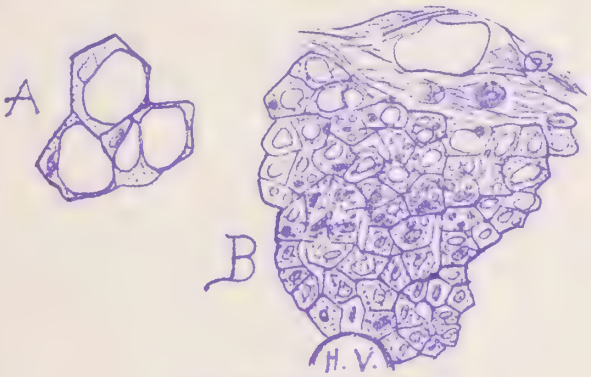



FIGURE 8.

A. Group of fatty cells showing large fat drops pushing nucleus and protoplasm to one side.

B. Portion of a lobule in fatty infiltration. Large fat drops in peripheral cells. Smaller ones towards centre. Normal cells about central vein.

cell pushing the protoplasm and the nucleus, which still stains well, to one side, where it appears flattened between the drop of oil and the cell membrane.

Even though fatty infiltration may at times be very extreme if the cause be removed the organ will, provided the process be not too advanced, return to the normal. This is a point which marks an important difference between this condition and the other fatty change to which the liver is subject, i.e. fatty degeneration.

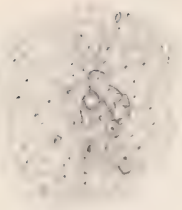
# FATTY DEGENERATION.

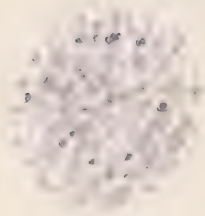
DEFINITION:- A diseased condition of the liver in which the protoplasm of the liver cells, by a process of degeneration, becomes converted into fat.

ETIOLOGY:- Fatty degeneration is found in the livers of those who have died from such wasting diseases as tuberculosis, cancer, Addison's disease, Anaemia and the like. It is also the result of certain poisons, notably phosphorus and arsenic. It is the sequel of cloudy swelling and occurs often in those who have died of continued fevers.

It is impossible to make a sharp distinction between fatty degeneration and fatty infiltration although the two processes are distinct and differ in many respects, both when viewed with the naked eye, and when studied under the microscope. In the livers in which fat is frequently found normally, it is often practically impossible to determine whether a given case is one of infiltration or degeneration. The process differs from infiltration in that it is strictly one of death, of degeneration, of the liver cells. The fat seen within the cells is the resultant product of the death of the cell protoplasm and not an increased production of fat, or storage of fat within these cells. While from fatty infiltration the liver may recover if the cause is removed in time, from fatty degeneration there is no recovery. The protoplasm once changed into fat cannot be reconverted into protoplasm. The process ends finally in the complete death of the cell which becomes converted into a mass of fatty granular detritus.

PATHOLOGICAL ANATOMY:- The liver is reduced in size. For this reason it is sometimes spoken of as the atrophied fatty liver. When the wasting is pronounced the capsule is found to be dull, more or less opaque and wrinkled.





On section the color of the organ is yellowish, or yellowish brown and, as in fatty infiltration, this changes can be frequently seen about the periphery of the lobules at first. The tissue is usually pale, friable and less firm in consistency than normal. Oil drops can be scraped from the surfaces.

PATHOLOGICAL HISTOLOGY:- Under the low power one can see that the livers lobules are fairly well outlined by a zone of fatty cells about their periphery. As a general thing these fat droplets are smaller than those seen in fatty infiltration, though in some cases, especially in phosphorus poisoning they are quite large.

Under the high power one sees that the droplets of fat are largest in the cells of the extreme periphery of the lobules. Here the livers cells are found to be shrunken and angular. In many places they have entirely degenerated, leaving nothing but a small mass of fatty granules. In other places cells are seen which contain several good sized globules of oil and whose protoplasm is very distinctly granular and stains poorly. No nucleus can be demonstrated in these cells. As we pass further toward the centre of the lobule the droplets become smaller and smaller and we meet with fewer degenerated cells. Here and there cells are seen which stain well and in which a nucleus can be found. Finally as we approach the central area, more and more healthy cells are met with, or cells showing only cloudy swelling, until just about the intralobular vein there is found a narrow zone of cells in which no fatty changes are seen When the process is very advanced however the fatty changes occur throughout the lobules. The capillaries in such a liver are usually clearly defined and easily seen.

PHOSPHORUS POISONING:- Where the fatty degeneration is acute, as in cases of phosphorus poisoning, the liver is not diminished in size; it is pale usually everywhere, and may

show small hemorrhages in places. Here and there the tissue will have a very decided yellow color due to the rupture of a small bile duct. The drops of oil found within the cells are larger and more nearly like those seen in cases of fatty infiltration. This is because the process has been so rapid that there has been no time for absorption of the fat to occur.

# Amyloid Disease of the Liver.

Synonyms:- "Waxy Liver", "Bacony Liver," "Lardaceous Liver," "Albuminoid Liver."

Definition:- A disease of the liver characterised by the presence about the cells and in the walls of the vessels of the organ, of "a peculiar, foreign, solid substance having a composition identical with albumen." (Hamilton).

Etiology:- Amyloid disease of the liver is found associated with conditions of extensive suppuration, with pulmonary tuberculosis, caries, chronic syphilis and a few other diseases.

Pathological Anatomy:- The liver affected with amyloid is increased in size and weight. This increase, in some cases is very marked, the organ being nearly double its normal dimensions. Its specific gravity is also increased. The tissue pits readily on pressure, but the depressions rapidly vanish when the pressure is removed, showing that it is quite elastic. Owing to the increased size of the liver its capsule is stretched, thin and translucent.

The color of the organ is paler than normal owing to the fact that the vessels contain very little blood. The cut section is of a greyish-yellow color and may in places show the characteristic appearance of fatty infiltration with which amyloid disease is frequently associated. The cut surface is also described as having "a peculiar dry lustre, comparable to that of a wax model".

Pathological Histology:- When a typical case is examined with the microscope, the wax like substance is seen to be deposited in the middle zone of the lobule. Here it is sometimes difficult to make out any liver cells, their place being

taken by the amyloid material. In the portal and hepatic zones the cells appear normal or slightly fatty. The amyloid substance which has been deposited around the cells in the middle zone presses upon them to such an extent that in time they atrophy and disappear. Examination of the small arteries and capillaries shows that there is here also an abundant deposit of the same substance, and it is probable that these structures are the first to become affected.

A diffuse form of this disease is sometimes met with in which the entire capillary net-work has become simultaneously affected. The wax-like material in these cases is deposited only about the vessels and not about the cells, which however are somewhat atrophied because of the pressure brought to bear upon them. The atrophy is much less marked than in the other form of the disease

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For the methods of staining the Amyloid Material see Appendix.
For an account of the nature of the Amyloid Change see Lecture Notes on "Amyloid Degeneration."

# Cirrhosis of the Liver

Definition - A chronic disease of the liver characterised by an overgrowth of the connective tissue elements of the organ and a destruction of liver cells (Chronic Interstitial Hepatitis)

Etiology. - The chief factors in inducing these changes in the liver may be summed up in brief as follows

1 Alcohol. This is probably by far the most important agent in producing cirrhosis. The undue use in a large number of cases must undoubtedly be considered the direct cause

2 Syphilis. This disease ranks next to alcohol as a causative agent both the congenital and the acquired form frequently being associated with a distinct variety of cirrhosis

3. The habitual use of highly seasoned food.

4 Long standing chronic passive congestion of the liver. This is however doubted by some authorities, (i e Hamilton).

5 Prolonged malarial poisoning

6. Scarlet and typhoid fevers

7. Rickets

8. Anthracosis. With pronounced cases of this disease there has been noted, especially by Welch, a form of cirrhosis associated with a considerable coal dust pigment in the liver.

The disease is essentially one of middle and advanced life though a certain number of cases occur in children

Varieties. - Two main forms are met with known respectively as the Atrophic and the Hypertrophic. Minor varieties are also found, chief among which may be mentioned Fatty Cirrhosis and Perihepatitis

# ATROPHIC CIRRHOSIS.

SYNONYMS:- "Hobnail" Liver, "Small Cirrhotic" Liver. "Course Cirrhosis", "Gin-drinkers" Liver, "Polylobular Cirrhosis." "Alcoholic Cirrhosis."

PATHOLOGICAL ANATOMY:- As the name atrophic indicates the liver is found at autopsy to be considerably reduced in size, frequently not being of more than one half its normal proportions, its weight also being correspondingly diminished. The organ is not infrequently deformed, and its surface presents numerous elevations and depressions which vary considerably in size and shape, giving to this condition the name of "Hobnail Liver." The capsule appears to be thickened, especially at the bases of the depressions just mentioned. The organ is firm to the touch, is tough, tears with difficulty, is quite inelastic and feels very much like a piece of stalked leather. On section the color is usually distinctly yellowish, (from which the name Cirrhosis -yellow) though occasionally it is of a reddish hue.

The liver cuts with comparative difficulty, and upon closely examining the cut surface one can see that the tissue is everywhere beset with connective tissue, which, running here and there cuts up the organ into many irregular sized masses of liver cells. If the section has passed through one of the superficial depressions, bands of fibrous tissue can be seen running from its base into the liver substance in all directions. It is owing to the contractions of these bands that the superficial pits are due.

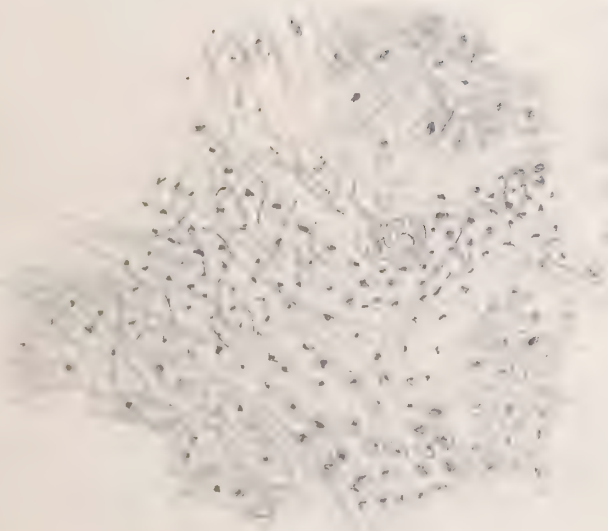
In the very earliest stages of this condition the liver may be slightly increased in size, due probably to the hyperaemia present at this time.

PATHOLOGICAL HISTOLOGY:- When carefully studied under the microscope it will be seen that the superficial layer of the capsule is very little, if any thickened. The deep layer

Liver

Atrophic Cirrhosis

Portion of lobule under low power and also fibrous connective tissue between lobules and also portal system in fibrous band.



however, that portion which is continuous with Glisson's capsule will be found to be many times its normal thickness, especially at the bases of the superficial depressions, from which one can now clearly see bands of fibrous tissue running into the liver substance. These soon divide, subdivide and reunite, forming in this way a coarse net-work in the meshes of which lie masses of liver cells of varying size. These main bands of cicatricial like tissue are well defined and only occasionally can be seen to send off fine fibrils between the cells of the lobules. This is held by many to be a distinctive character istic of this form of cirrhosis. About all the portal systems also the connective tissue is increased and from these it runs in broad bands into the liver substance in every direction.

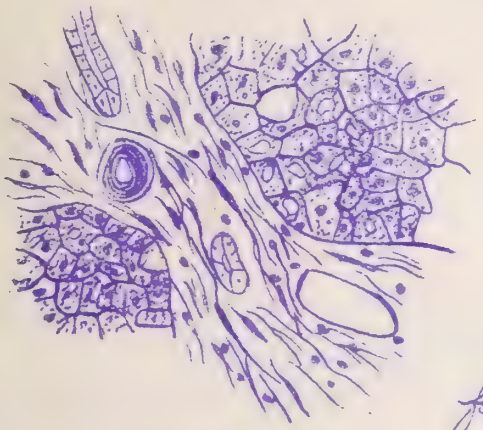


FIGURE 9.
Atrophic Cirrhosis
Showing band of connective tissue cutting up the liver substance. Liver cells slightly fatty.

If we examine, under the high power the margins of these well defined bundles of connective tissue, we will see that certain changes have been going on in the liver-cells which lie next them. These changes are all atrophic and result in the ultimate destruction of these cells. As the fibrous tissue increases and invades successively more and more of the parenchyma, numbers of liver cells are, as it were shaved off from the lobule by the advancing growth and become enveloped in its meshes. Here after a time, owing to the constant pressure which is exerted upon them, they soon begin to decrease in size until finally they present no resemblance to a liver cell whatever and lie some distance from the edge of the lobule. They ultimately entirely degenerate and disappear. Farther within the lobules the cells, in many cases will be found to be in a state of fatty infiltration. Other changes can sometimes be made out with the high power. These consist

chiefly in the enlargement of certain liver cells and of their nuclei which then proceed to divide and to subdivide, becoming in this way smaller and smaller. On the other hand in the majority of cases, instead of this proliferation of liver cells, the nuclei of many of the cells will be seen to have enlarged somewhat and then instead of proceeding to divide, retrogressive changes set it resulting in the formation of a vacuole within the nucleus and the final degeneration of the cell.

ORIGIN OF THE CONDITION:- For many years it was thought that the process of connective tissue increase in these cases of cirrhosis was a primary one, which started because of some action of the supposed cause. This increase in the fibrous elements of the organ lead naturally to the destruction of the proper cells of the part, as has just been described, by pressure. Of late however, it would seem that this view must be modified somewhat, and although we must recognise that when the fibrous tissue has once formed, it will produce an atrophy of liver cells because of its constant pressure, we now look upon the increase in connective tissue as a secondary process, following upon some primary disease of the liver cells, causing them to degenerate, their place being taken by a new growth of connective tissue in the same way that in an extensive wound, which the proper tissue of the part is unable to repair, this tissue enters and fills up the space.

Whether the process be primary or secondary, however, the new tissue is derived from that which is already in existence in the portal systems and supporting structure of the organ, although Flesch, Hamilton and some others have described the formation of connective tissue from the proper liver cells by the process of enlargement, division and subdivision already mentioned as being noticed in a very small number of cases.

# CIRRHOSIS.

## HYPERTROPHIC CIRRHOSIS.

SYNONYMS: - "Large Cirrhotic Liver." "Fine Cirrhosis." "Biliary Cirrhosis." "Monolobular Cirrhosis." "Granular Liver." "Hyperplastic Fibroid Induration."

PATHOLOGICAL ANATOMY: - The organ is enlarged, at times weighing as much as 7 to 9 pounds. This enlargement has led to the term Hypertrophic Cirrhosis, which though it is in constant use, is in fact misapplied, because on microscopic examination, as we shall see in the next paragraph, the proper liver cells are found to be in a state of atrophy and degeneration, while the enlargement of the organ is due to an increase in the interstitial tissue more rapid than the destruction of the liver cells.

The surface of the liver, which in the early stages is smooth, becomes, as the disease advances and the new formed fibrous tissue shrinks, somewhat finely granular in appearance. The liver tissue is firm, dense, even hard at times and tears with ease. In the majority of cases the surface shows yellowish or green-ish patches, which when the organ is sectioned can also be seen scattered through its substance. This discoloration is due to the absorption of bile by the liver cells. Here and there throughout the liver substance run numerous fine bands of connective tissue which cut it up into small lobular shaped masses. The capsule is very little, if any, thickened. The fibrous tissue in the portal

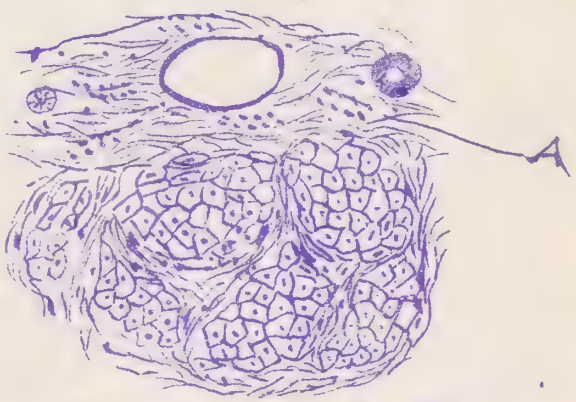



FIGURE 10. - Hypertrophic Cirrhosis

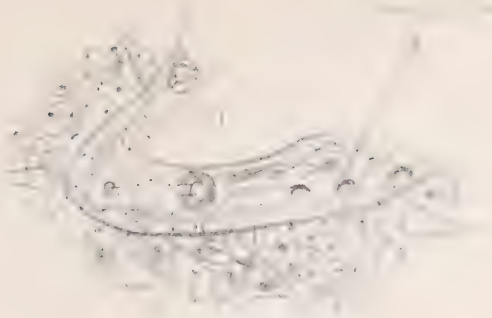
Portion of a lobule and portal system showing the increase in the bile ducts (A) and the invasion of the lobule by fine bands of connective tissue

systems is increased in amount, and occasionally the portal veins seem to be dilated.

PATHOLOGICAL HISTOLOGY: When examined microscopically fine bands of connective tissue are seen to traverse the section in all directions. These, it will be noted, lie about the periphery of the lobule surrounding them on all sides. This condition is made out with especial ease in the earliest stages, in some cases the lobules being so well marked out that the section resembles closely that of a pig's liver. The new growth of fibrous tissue, as in Atrophic Cirrhosis, begins in the portal systems, chiefly about the bile ducts, and from here grows out into the interstices between the lobules from these main bundles, which are much smaller than in the atrophic variety, smaller bands pass into the lobules between the liver cells, and by the pressure which they exert cause them to atrophy and degenerate. Like atrophic cirrhosis this variety advances by exacerbations, and if the section be from a liver in which the process is actively advancing one will find in and about the portal systems and the larger bands which run between the lobules, a certain amount of proliferation of connective tissue cells, the young cells appearing as masses of small round bodies with deeply staining nuclei.

In cases where the disease is, so to speak quiescent, these masses will not be seen, the new cells having grown out into fully developed connective tissue corpuscles.

BILE DUCT STRUCTURES:- Embedded in the new-formed connective tissue, especially in those cases which are somewhat advanced, there will be found numerous double rows of nuclei. These represent minute bile duct like structures. Sometimes they can be seen following the finer bands of fibrous tissue into the substance of the lobule, and again within the lobule one will occasionally see a few similar structures. According to Hamilton these are formed in the following manner. Fine



bands of connective tissue growing into the lobule surround double rows of liver cells. When this happens the cells so enclosed become smaller and smaller until nothing is left of them but their nuclei and a little protoplasm. When the specimen is injected through the biliary system these structures are found to be connected with the main bile ducts. From this fact he argues that the rows of liver cells in the normal liver are to be regarded as essentially expansions of the bile duct epithelium, and that in this form of cirrhosis they revert to their bile-duct-like embryonic condition. When the disease becomes very far advanced these structures, to a certain extent disappear. Occasionally cases of hypertrophic cirrhosis are met with in which there appears to be no new formation of bile ducts.

## FATTY CIRRHOSIS.

In both the Atrophic and Hypertrophic variety we frequently find that the cells of the liver are in a state of fatty infiltration. In cases of typical fatty cirrhosis however the organ is not reduced in size, but is somewhat larger than normal. In consistency and color it very much resembles an ordinary fatty liver. The capsule is smooth and glistening, though occasionally it may be slightly granular in appearance. The liver is firm and cuts with some difficulty, and when examined microscopically, there is seen to be not only an extensive deposit of fat within the liver cells, but also an extensive formation of connective tissue throughout the organ

# CIRRHOSIS.

| ATROPHIC | HYPERTROPHIC. |
|---|---|
| 1. The liver is decreased in size | The liver is increased in size. |
| 2. The surface of the organ is roughened with projections of considerable size. The capsule is quite thick. | The surface of the organ is but slightly roughened, is rather granular in appearance. The capsule is rarely thickened. |
| 3. There is little or no attempt at new bile duct formation. | New bile duct formation is characteristics of this condition. |
| 4. There is little or no bile staining of the tissues. | The tissues are frequently discolored by bile. |
| 5. The bands of connective tissue are coarse and enclose various sized areas of liver cells | The bands of connective tissue are fine and surround individual lobules, sending finer bands into them. |
| 6. The increase of connective tissue in the portal canals by compressing the smaller branches of the portal veins causes a congestion of the portal system in the abdomen and ascites results. | The new formed connective tissue compresses the bile ducts more than it does the portal veins and therefore causes a retention of bile and a consequent jaundice but not ascites. |

# CIRRHOSIS.v

## PERIHEPATITIS.

SYNONYM:- "Glissonian Cirrhosis"

In this condition the liver is greatly reduced in size and before death can not be diagnosed from the ordinary

atrophic variety of the disease. At autopsy not only is the reduction in size notable, but there may also be considerable alteration in shape. Two forms are met with. In one the capsule of the organ is very much thickened, tough and of a cartilaginous consistency. It is usually very firm and greyish-white in color. When this is stripped off, which can be done easily the liver substance beneath looks normal, but when examined carefully generally shows some cirrhotic changes.

In the other form the capsule is very little altered, the increase in connective tissue being confined to the portal systems chiefly, and showing little tendency to involve the liver substance.

The over-use of alcohol is the most common cause.

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## ACUTE INTERSTITIAL HEPATITIS.

DEFINITION:- An acute inflammatory condition of the liver affecting particularly the fibrous tissue elements of the organ.

ETIOLOGY:- Acute interstitial hepatitis is most frequently associated with cases of small-pox, scarlet fever, measles, typhoid fever, or the other specific fevers, and is found also in the early stages and during the exacerbations of the various forms of cirrhosis.

PATHOLOGICAL ANATOMY:- In size, the liver, if altered at all is slightly increased owing to the congestion which is always present. Upon section of the organ blood escapes freely, and when the cut surface is examined closely small grayish or red translucent spots can be seen here and there. Besides these changes there is nothing of note to be observed.

PATHOLOGICAL HISTOLOGY:- Cloudy swelling (see back) is always found in this condition, especially if the patient has died of one of the specific fevers. In the portal canals particularly, and at times in the fissures between the lobules where there is a slight amount of connective tissue, numerous small round cells are seen. These are chiefly the result of a proliferation of the connective tissue, though some of the cells may be leucocytes which have migrated from the vessels. It is these small masses of cells which give rise to the gray gelatinous points noted in the liver at autopsy.

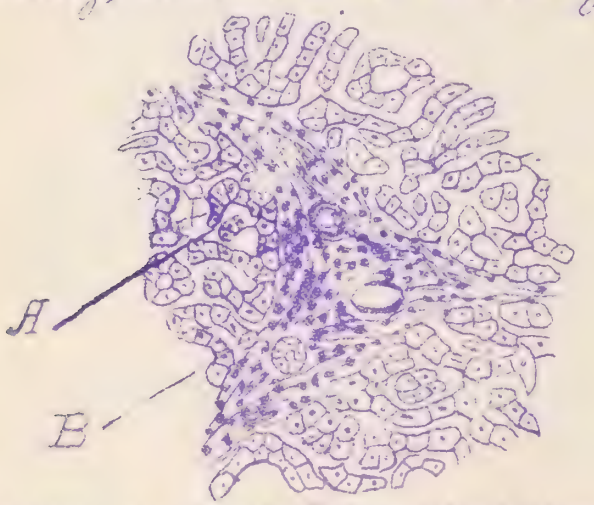



FIGURE 11. Acute Interstitial Hepatitis

A. Normal liver tissue. B. Interlobular connective tissue infiltrated with small round cells.

We have in these cases, evidently the beginning of a cirrhotic process. Most of the small cells with deeply staining nuclei are young proliferated tissue cells which in time develope into fully formed fibrous tissue and bring about the changes so characteristic of cirrhosis.

A similar picture is seen in cases of early acute abcess formation; but here the cells instead of going on to the formation of connective tissue, degenerate, probably because of the presence of pyogenic (pus producing) organisms.

# SYPHILITIC DISEASE OF THE LIVER.

Syphilis affects the liver chiefly in two ways which differ markedly from each other. These will be described under

I. Congenital Syphilitic Cirrhosis; and
II. Syphilitic Gumma of the liver.

## CONGENITAL SYPHILITIC CIRRHOSIS.

This form of the disease is met with in still-born syphilitic infants and also in those who have died in childhood with other signs of congenital syphilis about them. Some cases are on record in which, although there were no symptoms of syphilis, the autopsy disclosed this condition of the liver.

PATHOLOGICAL ANATOMY:- At autopsy the liver is either noted as normal in size, or as slightly enlarged. In fact, so slight may be the gross changes, that without further examination the organ might be considered normal. In other cases, beside the slight enlargement the surface may be slightly discolored, or there may be a few nodular projections here and there. In consistency the tissue is firm and tough and in color usually somewhat pale in spots.

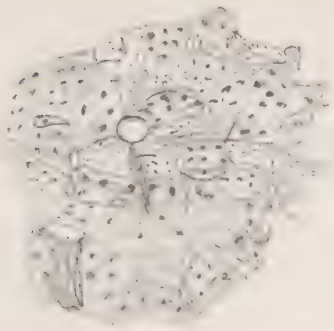
On section in mild cases no changes may be noted, while in others it will be difficult to make out in places, anything of the normal structure.

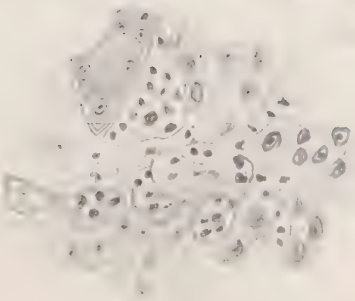
PATHOLOGICAL HISTOLOGY:- When examined microscopically very definite changes can be seen. In the milder grades of the disease the liver tissue will be found to be beset with numerous small round cells. The interlobular tissue is usually the most affected, though infiltrated patches lying within the lobules are not uncommon. The aggregation of these small round cells is also seen within and about the

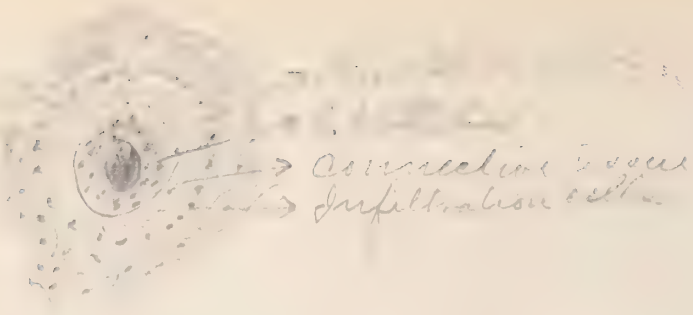
capillaries. When the disease is more grave further destructive changes are found. The whole organ or numerous areas throughout its substance are seen to be the seat of new-formed connective tissue. This is not confined to the portal systems, nor does it extend in well defined bands, as in the varieties of cirrhosis already described, but follows the capillary net work of the lobules throughout. The liver cells as a consequence of this are found to be separated one from another by fine translucent fibrous tissue and on account of the pressure exerted by this to have assumed all sorts of shapes and sizes. They are in fact in a state of extreme atrophy and degeneration. The new formed tissue does not look like that seen in the ordinary forms of cirrhosis, but is of a finer texture and more translucent. It arises not only from the pre-existing connective tissue of the organ, but from the endothelial cells which line the capillaries and lymphatic spaces as well. On account of the proliferation of these cells we have the small round celled infiltration noted in the earlier stages and ultimately, when the new tissue is fully developed, an occlusion of many of the capillaries (endarteritis obliterans) and lymph spaces which lie at the margins of the lobules and extend into them between the liver cells and interlobular capillaries

## Syphilitic Gumma of the Liver.

Syphilitic gummata may occur either as an evidence of congenital syphilis, or as a tertiary lesion of the acquired disease. Like syphilitic disease elsewhere all the lesions tend to the production of new connective tissue in which subsequently degenerative changes take places. Gummatous hepatitis, as this condition is sometimes called, is therefore closely allied to the diffuse congenital cirrhosis just described. In the present form however the new fibrous tissue is much coarser in quality and here and there tends to fall into gummatous decay.






→ Connective tissue
→ Infiltration cells

PATHOLOGICAL ANATOMY:- The liver affected with this disease is very frequently much altered in shape and size, so much so at times that all resemblance to the normal organ is lost. More frequently however the liver is but moderately distorted and presents here and there pronounced ridges and depressions, or pale depressed scar-like notches.

The capsule is usually thickened and shows evidences of previous inflammation in numerous adhesions to adjacent parts which are either old and fibrous or still fibrinous and easily broken.

Upon section of the organ, in many places wide bands of cicitricial-like tissue can be seen running in from the capsule, and scattered throughout the substance of the liver there will be found nodules of varying size. If the disease be in an early stage these will be greyish in color and pale, but when older they may be described as "pale yellowish, cheese like nodules of irregular outline, surrounded by a fibrous zone, the outer edge of which loses itself in the lobular tissue, the lobules dwindling gradually in its grasp. This fibrous zone is never very broad; the cheesy centre varies in consistency from a gristle-like toughness to a pulpy softness; it is sometimes mortar like from calcareous change". (Wilkes.) The deformity sometimes noted in the organ is produced by the gradual healing of these gummata.

PATHOLOGICAL HISTOLOGY:- It will be best to describe the minute anatomy of the young developing gummata and of the older gummatous masses separately.

The younger gummata are chiefly found in the livers of syphilitic children, either still-born, or dead of the disease, or of some intercurrent affection.

On examining a nodule taken from a liver of this kind, it is seen that the growth is situated in newly formed intra-lobular fibrous tissue; in other words that the formation of a gummd is preceded by a syphilitic cirrhosis identical with that

already described (see book); at certain points the development of fibrous tissue has taken place to such an extent that there are numerous strongly marked fibrous bands interlacing the lobules and subdividing up the liver tissue. These fibrous bands are highly vascular, vessels in all stages of development being seen in their substance. In the fibrous band and around the vessels the first trace of the developing gumma appears in the form of deeply stained embryonic cells (small round cells) forming a ring which gradually increases in diameter; as this extends peripherally, the cells near the centre become angular granular, atrophied and with their nuclei stain poorly. (Woodhead).

The gumma just described is an actively growing mass of connective tissue. All the important changes however are not confined to this mass alone. If we examine the vessels in the neighborhood we will find that their coats have become thickened, briefly through an increase in the thickness of the intima, or inner coat. On account of this thickened condition the lumena of the vessels are at times completely obliterated. Where they are still pervious so far as the thickening is concerned, they will in many instances be found to be plugged with a fibrin coagula.

In older gummata, under the high power the changes which have taken place can be made out with greater distinctness. These may be briefly summed up as follows.

1. In the centre of the mass nothing but granular, degenerating, detritic, cells.
2. Surrounding these a zone of large cells similar to the cells described as "epithelioid" in speaking of the structure of a tubercle. They are probably the same.
3. About the margins of the mass a certain amount of fine connective tissue.
4. Outside of this a zone of small round-celled infiltration.

-Gummata which have reached a certain size almost invariably undergo caseation which is probably due both to the specific action of the syphilitic poisons and to the obliterating end-arteries which is going on in the vessels, by which the blood supply is gradually cut off.

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## THE LIVER LESIONS IN TYPHOID FEVER

Although the chief lesion of typhoid fever is found in the intestines (see Typhoid Ulcer of the Intestines), other parts show certain changes and the liver is usually affected in the following way.

PATHOLOGICAL ANATOMY:- In the early stages of the disease it is hyperaemic, and in a large proportion of cases is somewhat swollen. In color it is paler than normal and the cut section looks granular and turbid. Scattered throughout the substance of the organ may be seen numbers of small spots, greyish in color and about the size of a pin's head. These resemble somewhat the small tubercles found in the liver. In a small percentage of cases abscesses may be found, and acute yellow atrophy is extremely rarely met with.

PATHOLOGICAL HISTOLOGY:- Beneath the microscope the small greyish spots just mentioned are found to lie chiefly in the zone of the hepatic artery, though in advanced cases they spread rapidly and are found also in the zone of the portal vein (peripheral zone) as well. These minute nodules are composed of necrotic liver cells in all stages of disintegration. It has been supposed that they were caused by the impaction of masses of bacteria within the lobular capillaries, of the intermediate and peripheral zones, but Reed working under Welch, has made a careful study of this lesion and can find no relation between these areas and the bacterial infection. Hamilton also states that he has been unable to detect any bacteria within the necrotic nodules.

Ultimately the necrotic cells fall to pieces leaving only a small circumscribed mass of granular and fatty detritus. Aside from these localized lesions the liver cells show wide spread cloudy swelling, and in many cases with marked fatty degenerations (See back, Cloudy Swelling and Fatty Degeneration.)

# LEUCOCYTHAEMIA.

SYNONYM:- Leukaemia.

DEFINITION:- "An affection characterised by persistent increase in the white blood corpuscles, associated with enlargement either alone or together, of the spleen, lymphatic glands, or bone marrow" (Osler.)

ETIOLOGY:- Nothing is known as to the cause of this condition. (For further details see any work on the practice of Medicine.)

While the chief pathological conditions are to be found in the parts above mentioned the liver also shows certain changes which are worthy of mention

PATHOLOGICAL ANATOMY:- This organ is usually enlarged. In one case described by Welch it weighed over thirteen pounds. In consistency it is firm; its surface is smooth and pale. Beneath the capsule there may be small bright haemorrhages. When the organ is cut, the surface is seen to be paler than normal. This is most marked in the interlobular fissures giving to the section the appearance of a "network, or irregular veining of pearly white color.

PATHOLOGICAL HISTOLOGY:- When studied microscopically the columns of liver cells are seen to be widely separated "by leucocytes, which are partly within and partly outside the lobular capillaries." These leucocytes appear as small round cells with deeply staining nuclei. Sometimes they are found aggregated in little clumps, most frequently in the peripheral zone, and when occurring in this manner they obscure the liver cells. When studied with the high power a few delicate threads of fibrin may be seen in places.

In some case pigmentation of the liver cells is found which, though it differs from the pigment found in cases of passive congestion is nevertheless ferruginous in character and is probably derived from the blood

# TUBERCULOSIS OF THE LIVER.

Tuberculosis affects the liver in two different ways, namely as a miliary tuberculosis and as a tubercular perihepatitis; this is to say that it affects two different parts, the liver substance and the capsule of Gleason. Frequently the one condition is present to the entire exclusion of the other, and the former is much more common than the latter.

## MILIARY TUBERCULOSIS.

PATHOLOGICAL ANATOMY. – At autopsy there are, in the majority of cases no gross evidences of the disease. In other cases upon section one may see small greyish nodules scattered throughout the liver substance, which are most numerous in the neighborhood of the capsule. When the disease is but little advanced these nodules may be so small as to be invisible but they ultimately increase in size, become non-vascular, caseous and bile-stained, so that upon section they now appear as yellowish points.

This form of the disease is in nearly every case secondary to advanced tuberculosis of the lungs, or is part of a general miliary tuberculosis. It rarely if ever occurs as a primary affection. From the tuberculous lesions in the lungs the tubercle bacilli enter the circulation and lodge in the capillaries of the liver lobules.

PATHOLOGICAL HISTOLOGY. – The liver is the best organ in the body on which to study the growth and development of tubercles as well as their structure when fully developed. Here the process is not complicated by secondary catarrhal changes such as are met with in the lung and which mask to so great an extent the normal process of growth.

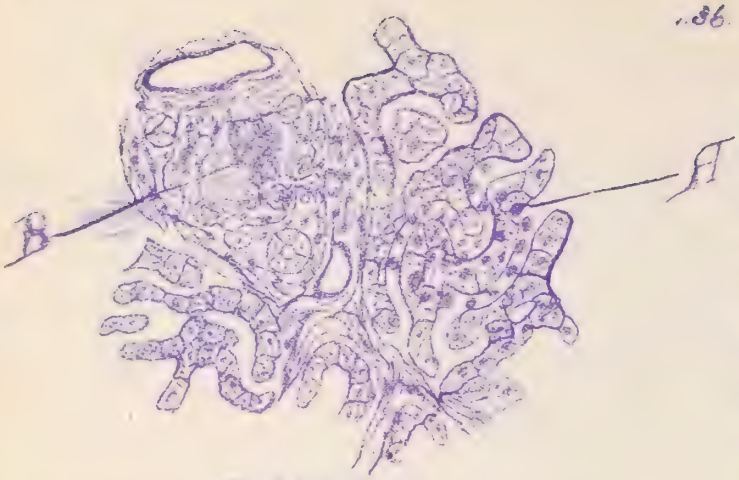



FIGURE 12.

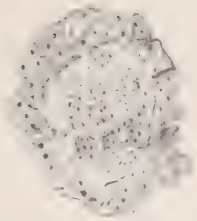
Miliary Tuberculosis of the Liver
A Young tubercle B Tubercle just beginning to become caseous

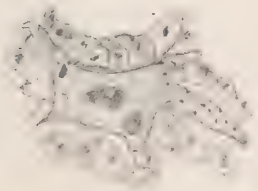
In its earliest stage the tubercle of the liver is seen to be simply a mass of small round cells with deeply staining nuclei, lying about the liver cells in a small circumscribed area. These cells are probably the result of proliferation of the fixed connective tissue cells and of the endothelial cells of the part, and their presence is due to the stimulating effect of the tubercle bacilli which have become lodged in the capillary vessels of the part. After a short time there is added to the little group a number of cells which, because of the inflammatory reaction set up by the bacilli, have migrated from the vessels. When examined in this stage with the high power we find that the tubercle is made up simply of migrated leucocytes or lymphoid cells and of proliferated cells, many of which have already increased somewhat in size and show a distinct vesicular nucleus, the epithelioid cells as they are called. The liver cells of the affected area at first show no changes but before long they become shrunken and granular and finally disappear.

FIGURE 13.

Types of Cells found in Tubercle
A Lymphoid B Epithelioid
C Giant Cells

If we examine a later stage, after time enough has elapsed for the tubercle to reach its full development and to begin to show retrogressive changes a different picture is presented. The nodule is now of considerable size and is sharply defined; at its center we find all the cells in a necrosed condition





appearing as a granular indefinite mass which is frequently bile-stained. About the margins there is a narrow zone in which the cells are dying, their protoplasm presenting a granular poorly stained appearance and their nuclei appearing as fine broken up fragments. (fragmentation of the nuclei). About the border we still find the same cells described as composing the young tubercle, and somewhere within the mass will be seen one or more large cells containing many nuclei, the giant cells. These are usually described as occupying the centre of the tubercle, but one finds them fully as often somewhere about the periphery of the mass. Running through the nodule and about its margins can be clearly seen fine delicate fibrils of connective tissue.

All liver cells have now entirely disappeared from the tubercle, and those about its borders appear flattened and granular. In some cases these older tubercles break down and form small cavities with sharply defined walls, containing a yellowish or greenish, bile-stained detritus.

These miliary tubercles most frequently spring up in the hepatic artery zone of the lobules and rapidly spread into that of the portal vein. They at times enclose small bile ducts which probably accounts for the bile staining so frequently seen.

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## TUBERCULAR PERIHEPATITIS.

This form of the disease is most frequently associated with tubercular peritonitis, though both may frequently be dependent upon pulmonary tuberculosis. The lesion is characterised by a considerable increase in the connective tissues of the organ, especially in the portal systems and in the capsule. This increase frequently takes on the appearance of an atrophic cirrhosis, and the tubercles instead of being found in the substance of the organ are confined to the

areas in which there is connective tissue, i.e. in the capsule and in the portal spaces. These tubercles like those in the substance of the organ are at first small greyish nodules, ultimately becoming larger, yellow and bile stained. After reaching a considerable size they break down and form cavities which contain a semi-fluid, greenish detritus. In very rare instances the entire organ may be honey-combed by these cavities, which vary in size from that of a pea to that of a pigeon's egg.

Histologically these tubercles are identical with those already described.

# ABCESS OF THE LIVER.

There are three chief forms of hepatic abcess one of which only we will consider here. These are abcesses due to tropical dysentary; the pyaemic abcess of the liver, and suppuration in connection with hydatid cysts.

## PYAEMIC ABCESSES.

ETIOLOGY:- These are quite rare unless there be suppuration somewhere within the area of distribution of the portal vein, such as putrid ulcer of the stomach or intestine, septic disease of the pelvic organs, etc. In these conditions there is absorption of septic material from the original seat of the disease by the branches of the portal vein which carry it immediately to the liver.

PATHOLOGICAL ANATOMY:- In consequence of this mode of infection the abcesses are multiple and widely distributed. When lying near the surface the tissue over them is less firm in consistency than normal and frequently fluctuation may be obtained. Upon section of the organ numerous cavities are met with scattered throughout its substance which vary in size from that of a large pin's head to that of a hen's egg. In some instances several of these cavities will have by their confluent forming a large faculated excavation. The walls are ragged and slouchy, and the contents are frequently foul in odor and of a greenish yellow color. The surrounding tissue is usually soggy and pale in color.

PATHOLOGICAL HISTOLOGY:- The walls of these abcesses are found to be infiltrated with numerous small round cells and these are also found in considerable numbers around the vessels in the immediate neighborhood. The edges

of the walls themselves as previously mentioned are ragged and portions of necrotic tissue, which takes the stain very poorly can be seen projecting into the cavity. The contents are seen to be made up of pus cells in all stages of necrosis and disintegration. If the specimen be stained for micro-organisms (See appendix) these will be found as bluish, or violet stained masses in the clots in the vessels and in the capillaries. The liver cells in the neighborhood of the abcess are granular and in some instances completely degenerated and ~~broken~~ down into a granular mass.

# Pigmentary Infiltration of the Liver.

Pigmentation, or pigmentary infiltration of the substance of the liver is a condition not infrequently met with. When it is considered that the circulation within the liver capillaries is very slow and that in consequence any foreign particles afloat in the blood are readily deposited it will be easily seen why this is so. If, as frequently happens, there are large numbers of disintegrated red corpuscles in the blood, these, or the colored iron compounds, which have been set free by the disintegration soon come to be deposited within the liver. These first lodge in the capillaries of the interlobular connective tissue, and later in the vessels of the peripheral zone of the lobules. After a time they may escape from the vessels and appear in the connective tissue and within the liver cells themselves. The condition though common is not usually so extensive as to interfere with the functioning of the organ, though occasionally such masses of pigment are met with that the liver cells are obscured in places. The pigments derived from the blood are either yellow, brown or black, and the color imparted to the tissue varies accordingly.

At times the pigmentation will be so regular as to outline the lobules very well.

Biliary pigmentation is of a different nature and being distributed in an entirely different manner will be found in different situations, generally extensively distributed within the liver cells throughout the lobules. The biliary pigmentation may be present to quite a marked degree in the liver, even though during life, the patient was not jaundiced. The color of the bile pigment is usually of a bright yellow and occurs as very fine granules within the liver cells.

# THE SPLEEN.

For a full account of the normal anatomy of the spleen read Poirsol's or Schafer's Histology, or Gray's or Quain's Anatomy.

## AMYLOID DISEASE.

VARIETIES - The Spleen is more often affected with amyloid disease than any of the other organs. It here appears in two forms which are usually described as the "Sago-Spleen" and as the "Diffuse Waxy Spleen."

The first variety is by far the most common and will be first described. By staining these two varieties of this disease the normal structure of the spleen is quite clearly shown and for this reason amyloid disease is here described first among the few splenic diseases that we will study.

## SAGO-SPLEEN.

PATHOLOGICAL ANATOMY: - The organ is generally enlarged and may weigh as much as 40 ounces. In consequence of this enlargement, the capsule is somewhat stretched. In consistency the spleen is firm and on section the tissue is usually found to be dryer than normal and quite friable. On closely examining the section with the naked eye one can make out the Malpighian bodies with great ease. They appear much larger than normal and of a pearly yellowish color, that resembles the appearance of boiled grains of sago, (whence the name). These bodies are usually rendered more prominent because of the dark red color of the surrounding splenic tissue. If we apply a solution of iodine to the cut surface these Malpighian bodies take on a mahogany red color.

PATHOLOGICAL HISTOLOGY:- Both stained and unstained sections should be studied. In the unstained sections the Malpighian bodies which are the seat of the amyloid deposit are seen to be glistening, translucent, hyaline in appearance and of a yellow color. In the stained sections, when haematoxylon and eosin has been used, the same bodies have a peculiar bright pink color. All the lymphoid cells which compose these bodies normally have vanished and only here and there can one see a few scattered, elongated nuclei stained blue. In the centre of the mass the central artery can still be seen.

About these amyloid Malpighian bodies the splenic pulp at first sight appears to be unaltered. If however a section be stained with gentian-violet which gives to the waxy material a beautiful pink color, a few reddish lines and dots can be made out here and there. These are sections of small vessels in the walls of which waxy material exists. Under the high power these amyloid vessels can be very easily studied and it is seen that the foreign material is confined to their middle coats. Usually in this form of the disease one also finds a very slight deposit of the amyloid material in the walls of the splenic sinuses immediately about the affected Malpighian bodies.

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## DIFFUSE WAXY-SPLEEN.

PATHOLOGICAL ANATOMY.- This condition has been described by some writers as simply an advanced grade of the sago-spleen, but this is probably not the case as the waxy material is deposited in quite different localities.

As in the former case the organ is enlarged, though the enlargement is here usually more marked. In consistency it is quite firm and elastic and its edges are rounded. The cut section has a dry, glistening appearance, but no such

"sago-like bodies as have just been described can be seen. If iodine solution be poured upon the cut surface the general color becomes a mahogany red, while the Malpighian bodies can be seen as minute yellowish spots.

PATHOLOGICAL HISTOLOGY:- Unlike the sago-spleen we see in this condition, when the section has been properly stained, that the Malpighian bodies are quite normal in appearance. The splenic pulp is however altered, and upon closely examining this we see that the wax-like material has been deposited in the wall of the splenic sinuses. One also finds that the same amyloid material exists in the walls of the arteries and small vessels in the Malpighian bodies. If these bodies are in any way altered it is by fibroid change, or by atrophy the wax-like material being deposited in other parts. Upon examining the splenic sinuses with the high power one can frequently see, that not only has the amyloid material been deposited in their walls, but that the cells which line them are in many places in a state of fatty degeneration.

# CHRONIC PASSIVE CONGESTION OF THE SPLEEN

DEFINITION:- A condition of overfullness of the spleen with venous blood due to some interference with the outflow of blood from the organ.

ETIOLOGY:- Valvular lesions of the heart, cirrhosis of the liver, emphysema, and fibroid conditions of the lungs all are associated with congestion of the spleen. (Describe on the opposite page the way in which ordinary cirrhosis of the liver produces passive congestion of the spleen.)

PATHOLOGICAL ANATOMY:- The organ is usually somewhat enlarged. It is heavier than normal and is flabby in consistency. As a usual thing the capsule is thickened and in places may have a cartelaginous consistency. In color the spleen is dark red and when cut bleeds freely.

PATHOLOGICAL HISTOLOGY:- As the name of this condition would imply the chief thing noticed when the section is studied under the microscope is the great distention of all the sinuses with blood. Upon more carefully examining the specimen one finds that the trabeculae are thickened and more prominent than normal and that there is an evident increase in the connective tissue of the organ generally. The increase takes the form of fine fibrils of fibrous tissue running through the Malpighian bodies, which are less prominent than normal, and through and about the splenic sinuses. The cells which line these sinuses are also in many places seen to contain altered blood pigment, and occasionally one sees large leucocytes similar to those seen in passive congestion of the lung which seem to be loaded with small granules of a light brown pigment, which has been derived from the haemoglobin of broken down red blood corpuscles. About the vessels of the spleen in this condition there is also seen an increase in the connective tissue, and along the thickened trabeculae one can in places see small masses of small round cells very much like the masses of cells seen in granulation tissue. Within the thickened capsule and in some of the larger trabeculae one can make out a certain amount of hypertrophy of the muscle fibers.

# TUBERCULOSIS OF THE SPLEEN.

ETIOLOGY:- Tuberculosis of the spleen, as with tuberculosis of all other parts is the result of the action of the tubercle bacilli. We never find this organ alone affected, that is it never occurs in the spleen as a primary affection, but is always found associated with tuberculosis of other parts.

VARIETIES:- Occuring in the spleen, tuberculosis is found in two forms, the one a miliary variety and part of a general acute miliary tuberculosis, and the other an extensive caseous tuberculosis, usually occuring with chronic tuberculosis of the lungs.

PATHOLOGICAL ANATOMY:- In the miliary variety the organ is but slightly altered in size or consistency, and upon section one can see very minute greyish points scattered everywhere over the surface. These are most numerous in and just beneath the kapsule. Sometimes they are found to be slightly larger and to have a pin-point yellowish centre which indicates that caseation is beginning in these masses which are the minute miliary tubercles.

The second variety as has been mentioned is more chronic in character. Here upon section of the organ extensive yellowish, firm cheesy masses are found, with sharply defined borders about which in the comparatively healthy splenic tissue one can frequently make out small greyish spots, the second crop of young tubercles. In this condition the organ is usually diminished in size and quite hard.

PATHOLOGICAL HISTOLOGY:- Under the microscope the miliary tubercles of the first variety are found to differ in no respects from miliary tubercles already described in other parts and will therefore not be described again here. (Write one this of your description, and make a sketch of one of these miliary tubercles.)

# LEUCOCYTHEMIA OF THE SPLEEN.

DEFINITION:- A condition associated with the general disease Leucocythemia.

ETIOLOGY:- Unknown.

PATHOLOGICAL ANATOMY:- In this condition one meets with the largest spleens found at autopsy. So great this enlargement be that the organ may weigh several pounds. The increase in size occurs in all directions so that the shape of the organ is not lost. It is firm and tough and in color pale. Upon section the splenic tissue presents an indefinite appearance and is usually greyish in color with here and there a redder spot due to ~~some~~ small haemorrhage. The Malpighian bodies appear as grey points scattered over the surface.

PATHOLOGICAL HISTOLOGY:- Under the low power one can easily see that the trabeculae are thickened and the Malpighian bodies somewhat enlarged, though in most cases the latter are not sufficiently altered to attract attention. Everywhere throughout the section there are large numbers of round cells, some of which are hemorrhagic while others are much larger and are of different origin.

Under the high power the changes in the trabeculae and the Malpighian bodies are more easily made out. Occasionally one can distinguish a fibroid condition of the latter. In the splenic pulp however the most noticeable changes are met with. Here the pores are found to be distended and to be filled with cells, many of which are leucocytes, and many also larger cells which it can now be seen are derived from the cells which line these spaces. These lining cells are found to be swollen and enlarged and in many places to be attached to the walls by a pedicle, while others are found which have evidently just been detached. Most of these cells contain one nucleus, but some are found with two. In some cases they contain altered blood pigment. It will thus be seen that the changes in this condition chiefly occur in the pulp of the spleen and not in the Malpighian bodies as was once supposed.

In Pseudo-leucocythemia, or Hodgkins disease the chief changes occur in the Malpighian bodies and not in the splenic pulp, and the changes are thus not to be considered the same as is stated by some writers.

# THE PANCREAS.

## CIRRHOSIS OF THE PANCREAS.

This condition is stated by most authorities to be only very occasionally met with. It is probably of comparatively rare occurrence in any marked degree and upon what it depends is unknown. Those causes which are so prone to produce cirrhosis in other organs, such as the liver and kidney seem to affect this organ in a much less degree. Cirrhosis of the pancreas has of late been noted as occurring in a certain percentage of cases of diabetes. The present specimen is from a case of this kind

PATHOLOGICAL ANATOMY:- But little can be said of the gross appearance of the organ in this condition. In general it is firmer than normal and cuts and tears with comparative difficulty. In some instances the development of connective tissue may be so great as to cause the organ to lose much of its normal shape when the new formed tissue contracts. This was the case in the pancreas from which these specimens were taken.

PATHOLOGICAL HISTOLOGY:- Upon microscopic examination of an area in which the cirrhotic changes are marked one finds that there has been a great development of connective tissue which not only surrounds the groups of glandular acini in wide bands but also invades these groups, small and large bands cutting them up into all manner of shapes and sizes. Fine fibrils of connective tissue run in between the acini and the subsequent contraction cause considerable atrophy, so that in some places little if any of the normal structure of the pancreas can be made out.

# DIABETES AND LESIONS OF THE PANCREAS:-

That the pancreas is altered in many cases of diabetes is now a well known fact but no constant lesion has as yet been discovered. Cirrhosis, cancer, atrophy, cyst formation and abcess have all been described in connection with it. It is a well known fact that total removal of the pancreas causes diabetes.

Reasoning from these facts it has been argued that the organ produces, besides its well known products, some other body of the nature of a ferment which has the power when taken into the blood of breaking up sugar. This ferment being absent the sugar is not broken up and is excreted by the kidneys as such. If only a small portion of the pancreas be left in the body diabetes will not follow.

A

# LABORATORY MANUEL

of

# Pathological Anatomy

## SECOND PART.

## DISEASES OF THE LUNG

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# EMPHYSEMA

Two forms of Emphysema are recognised, the one known as Interstitial, and the other as Vesicular.

## INTERSTITIAL EMPHYSEMA

DEFINITION - This condition is characterised by the presence of air beneath the pleura and in the interlobular connective tissue.

Beneath the pleura little bubbles of air arrange themselves in rows like threads of beads, and in the interlobular septa they can be seen arranged in a somewhat similar manner. At times the pleura is dissected up over a considerable area forming in this way blebs of considerable size. Interstitial Emphysema should not be confounded with the post-mortem generation of gas in the interstitial tissue, which is not of unfrequent occurrence.

ETIOLOGY - The causes of interstitial emphysema are such as will give rise to rupture of pulmonary alveoli, forming a communication between them and the interstitial tissue, allowing in this way an escape of air from them into the tissues. Such a condition may be brought about by perforating wounds of the chest wall, or by injury to the lung by the end of a fractured rib. Violent coughing may lead to the same result, and for this reason it is sometimes found after cases of whooping-cough and bronchitis. The most favourite situation for this form of emphysema is the anterior borders of the upper lobes.

RESULTS. - Two rare results of this condition are possible.

1. Rupture of the sub-pleural blebs causing pneumothorax. This may or may not be accompanied by pleuritis.
2. Passage of the air beneath the pleura to the root of the lung, and thence into the tissues of the anterior mediastinum from where it passes upward into the subcutaneous tissue of the neck and chest, giving rise to subcutaneous emphysema.

# VESICULAR EMPHYSEMA

DEFINITION - A disease of the lungs characterised by the dilatation of the pulmonary alveoli and infundibula, frequently so great that rupture of their walls occurs, the condition being due "to persistent high intraalveolar pressure acting upon congenitally weak lung tissue." (Osler)

VARIETIES:- Two main varieties are met with, the difference between them being due to a difference in their causes. These are known as Ideopathic, or Hypertrophic and as Compensatory Emphysema

ETIOLOGY:- Of these, the first usually occurs as an independent affliction depending upon a variety of circumstances, while the second, as its name implies, is dependent upon the closure of a certain number of alveoli to air so that a greater amount is forced at each inspiration, into those which remain open. Compensatory Emphysema is sometimes spoken of as Vicarious Emphysema

Wherever there is consolidation of a portion of the lung there will be on the neighborhood areas of compensatory emphysema Such conditions as pneumonia, tuberculosis, closure of a bronchial tube, etc. which are associated with consolidation, or in which air is not allowed to enter a certain number of lobules, are always associated with a certain degree of this condition

Ideopathic Emphysema on the other hand is not dependent for its causation upon closure of areas of lung to the entrance of air. A large percentage of cases show a strong hereditary predisposition to this disease. Of etiological factors, the chief are, the playing of wind instruments, blowing glass, heavy lifting etc. It must not be forgotten however that in the majority of cases there must be at the same time a weakened condition of the pulmonary tissue, otherwise we would expect to find this condition in all who follow the above callings. Bronchitis also plays a part as a causative agent, because of the cough which accompanies it.

Cohnheim taught that the congenital pulmonary weakness was probably due to a defect in the development of the elastic fibers in the alveolar walls.

PATHOLOGICAL ANATOMY:- The chest is usually somewhat enlarged and is more nearly circular than normal, the condition being spoken of as "barrel chest" On opening the thorax the lung margins are frequently seen to overlap one another, at times concealing the pericardium The lungs themselves are larger than normal and occupy consequently more space in the chest cavity; they do not collapse when the thorax is opened, and when pitted with the finger do not resume their normal shape, their elasticity has therefore been impaired. In color they are lighter than normal, in many cases there being a notable absence of pigment, the condition being termed by Virchow "albinism" of the lung. When cut the knife passes through the tissue with difficulty. The organ has a "downy" feel and floats lightly on the surface of the water. The dilated alveoli can frequently be readily seen. When several of these have broken into a single space bullae of considerable size are formed

PATHOLOGICAL HISTOLOGY:- The most noticeable changes in the lung tissue are the increased size of the alveoli which are greatly distended, and the atrophy, the thinness of the alveolar walls. These in places are hardly more than threads in thickness, and in many places have ruptured. Where this rupture has occurred it will be seen that no haemorrhage has taken place as would be expected, the reason being that as the atrophy of the walls progressed the capillary net-work shared therein and the vessels became impervious to blood before rupture took place. Occasionally however a slight haemorrhage is met with. During the progress of atrophy and dilatation the alveolar epithelium usually undergoes a fatty degeneration and at times small droplets of fat may be seen within these cells.

SECONDARY EFFECTS OF EMPHYSEMA:- Owing to the extensive obliteration of the pulmonary capillaries there is obstruction to the flow of blood through the lungs which in time leads to an hypertrophy and subsequent dilitation of the right ventricle. When the condition is extreme and the cardiac changes not fully compensatory the result is general passive congestion of the abdominal organs.

Emphysema and tuberculosis rarely occur in the same lung, except in the case of localised vicarious emphysema dependent upon the tubercular consolidation.

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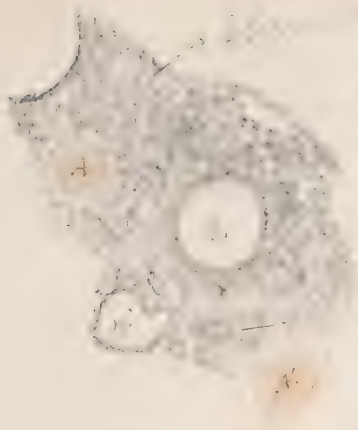
# COLLAPSE OF THE LUNG.

## ATELECTASIS.

DEFINITION:- A condition resulting when a part of a lung which has once functioned becomes airless

ETIOLOGY:- Collapse of the lungs may be met with under a variety of circumstances.

1. In those who before death have been unable to take full inspirations. In these cases the collapse is usually in the lower and posterior portions of the lungs.
2. In weak new-born children. Here the collapse is usually found in patches at the periphery, especially near the root and base of the lung.
3. In any case where pressure is brought to bear upon lung tissue as by aortic aneurism, spinal curvature, contraction of a thickened pleura, or by collections of air or extensive pleuritic effusions in the chest cavity. Under the latter condition the lung becomes compressed against the upper posterior part of the thoracic cavity.
4. In bronchio-pneumonia, where there are at times found lobular areas of collapse, probably due to plugs of mucous acting as ball valves at the mouths of some of the smaller


A
A'
A = Aorta
A+B' = Bronchial
A' = Oe
Esophagus

bronchial tubes, which allow the air to pass out of the portion of lung supplied by them, but which immediately fall back on inspiration and permit no air to pass into the lung beyond that point. It is also stated that the oxygen of the enclosed air is first absorbed by the blood, then the carbon dioxide and finally the nitrogen, the lung gradually shrinking to its foetal condition. (Birtherm)

PATHOLOGICAL ANATOMY:- In areas which have become collapsed the tissue is firm, tough and fleshy and of a reddish color, the alveoli are obliterated and their walls are usually congested because the part no longer expands and contracts with each respiration. Small portions of collapsed lung sink readily in water. At first the alveolar walls are simply in contact and should air again enter the tissue they would separate and the alveoli resume their normal shape. After a time however adhesion takes place, the tissue being transformed into a compact mass (carnification). It thus looses all resemblance to normal lung, the septa become thickened. Where the alveoli are not entirely obliterated they become filled with exfoliated cells, their epithelial lining being for the most part lost. In other cases there is an extensive formation of fibrous tissue. Where the atelectasis is due to the pressure exerted by an extensive pleuritic effusion the lung frequently becomes bound down by adhesions so that expansion is impossible. In these cases the lung is usually anaemic, and it may be oedematous. In cases of lobular collapse each of the atelectatic lobules appears as a leaden looking patch usually depressed somewhat below the surface of the surrounding lung. (See Broncho-pneumonia). The tissue is congested and there is emphysema of those lobules which are supplied by the other branch of the bronchial tube, or a portion of which has become plugged and so lead to the collapse.

PATHOLOGICAL HISTOLOGY:- Under the microscope the chief change to be seen is the absence of alveolar spaces.

The walls of the air vesicles are everywhere in apposition and the capillary vessels are seen to be engorged. In some cases there can be seen an increase in connective tissue in the collapsed areas.

The airless condition of the lungs in still-born children which closely resembles atelectasis, is spoken of as Apneumatosis.

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## PULMONARY OEDEMA.

DEFINITION - A condition of the lungs characterised by the filling of the alveoli, the bronchioles, and at times even the larger bronchi and the soaking of the interstitial tissue with fluid which has escaped from the capillaries.

ETIOLOGY :- Pulmonary Oedema may be due to a variety of causes. (1). It is very frequently associated with the death agony as a result of the gradual failure of the heart, probably in many cases appearing when the outflow of blood from the pulmonary veins is hindered by some obstruction which the right ventricle has not the power to overcome (Cohnheim), (Welch). (2). It is most frequently noted in cases of severe congestion as well as inflammatory conditions of the lungs, pulmonary tumors, hemorrhagic infarcts, etc. (3). At times it is found when all signs of a preexisting engorgement or of any of the conditions just mentioned are wanting. In this case the oedema is usually widely distributed and is probably referable to alterations in the capillary vessels due to the disease from which the patient has suffered. In this way is probably to be explained the oedema resulting from the inhalation of irritant gases as well as the typical form of this condition seen in those who have died from anaemia and Bright's disease.

PATHOLOGICAL ANATOMY:- The lungs are heavy; they frequently have a gelatinous appearance, pit on pressure and when cut large quantities of fluid escape which is either clear or if the oedema be dependent upon congestion, bloody. The favorite situation for pulmonary oedema is at the bases of the lungs, though as already mentioned it may exist throughout the entire organ.

PATHOLOGICAL HISTOLOGY:- Rarely do we have an opportunity to note the microscopic appearances in oedema because of the readiness with which the fluid escapes from the tissue when cut. If however, we have, before cutting the lung, tied off a small portion of the oedematous tissue and caused the serum within the alveoli, branchioles and interstitial tissue to be coagulated, we will be able to make out easily the great amount of such fluid within the lung, and the extent to which at times the alveoli are distended by it. Within the coagulated serum will be seen a varying number of swollen cells from the epithelial lining of the alveoli and branchioles, and a small number of leucocytes in some cases may be present.

# CHRONIC PASSIVE CONGESTION OF THE LUNGS.

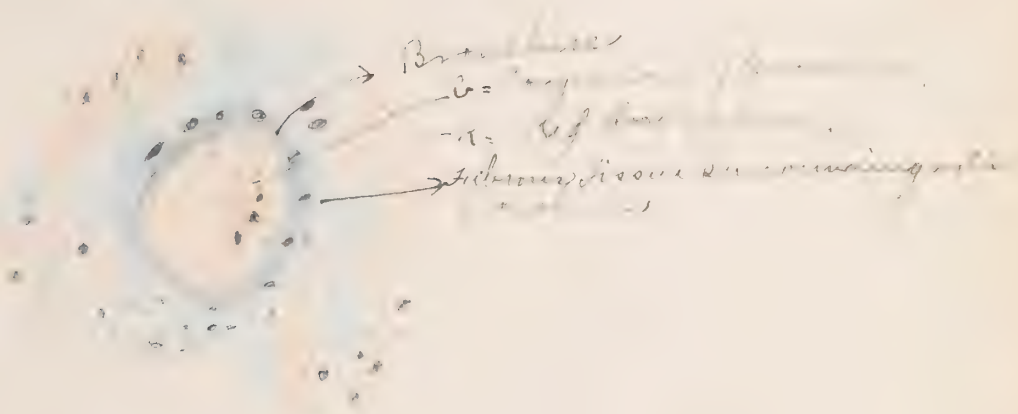
SYNONYMS: Venous congestion, Passive hyperæmia, Brown Oedema, Brown induration of the lung. Venous engorgment.

DEFINITION:- A condition of over-fulness of the pulmonary capillaries with venous blood due to some hindrance to the out-flow of blood from the lung.

ETIOLOGY- We may recognize two forms of passive congestion of the lung, the one mechanical and the other known as hypostatic. Mechanical obstruction to the outflow of blood from the lungs is most frequently caused by valvular lesions of the left side of the heart with broken compensation. Thus for example, if the mitral valve is incompetent, allowing at each systole a certain amount of blood to regurgitate into the left auricle the result will be that the inflowing blood from the pulmonary veins will find a part of the left auricle already occupied, and in consequence less blood will be able to enter from the lungs, causing a certain amount to remain in the pulmonary veins and ultimately damming back a like amount in the capillary system. The same effect is of course indirectly produced by lesions of the aortic valve. (Explain the production of passive congestion of the lungs by aortic stenosis). New growths or masses of enlarged lymphatic glands pressing upon the pulmonary veins will likewise interfere with the efflux of blood from the lungs and lead to passive congestion.

The causes of hypostatic congestion however are not so simple. This form of passive congestion is dependent upon an interference with one or more of the factors which maintain the circulation against the force of gravity. These factors may be briefly stated to be (1) the propelling power of the heart; (2) the so-called "aspiration of the thorax", the suction action of the chest, by which, at each inspiration the blood in the large venous trunks is as it were drawn into the heart, and (3) the movements of the skeletal muscles (in conjunction with the valves of the veins), their contractions helping to force the blood upward, while the venous valves prevent

Cronic-PASSIVE-CONGESTION
HELD-PAPER.



the blood to aberrate from falling back. If now we have any or all of these factors in the maintainance of the circulation impaired the tendency will be for the blood to flow less vigorously against the force of gravity, and consequently to stagnate in the most dependent portions of the body. For such conditions as protracted fevers, or wherever it becomes necessary for a person to occupy, for any length of time, a recumbent position, especially if the vital powers are diminished, we have not only a weakened heart action, but also shallower respirations and an entire absence of muscular movements, the result being that in such dependent portions of the body as the bases of the lungs, the under portions of the liver, kidneys and spleen, the calves of the legs, the bladder, &c. all become over-filled with the blood, not because of any mechanical obstruction to the outflow of blood from the parts but because of the weakened force of the circulation. (See Lecture Notes on Passive Hyperaemia).

PATHOLOGICAL ANATOMY:- As a rule the lungs are voluminous are of a deep reddish-brown color cut with difficulty and are heavier and firmer than the normal organ. In some cases the tissue bleeds freely when incised, while in others the organs are remarkably dry. At times there is considerable oedema. The color of the cut section is at first of a dark red, which however soon turns to a much brighter shade owing to the oxidation of the abundant haemoglobin. In advanced cases scattered over the cut surface there are irregular shaped patches of semi-solid lung tissue which have a rusty brown color and are dryer than the surrounding parts. Although as a rule the tissue will float in water occasionally one of these patches will sink. In this extreme form of passive congestion in which the tissue is almost airless the terms "Hypostatic Pneumonia" and "Splenization" have been applied.

PATHOLOGICAL HISTOLOGY:- When examined microscopically it is seen that (1) the capillary net work is exten-

evenly engorged with blood. One can frequently follow the twisting course of an individual branch for some distance and note that it rises from the alveolar wall in the shape of a loop and projects into the alveolar cavity for some distance. If the specimen has been hardened in formol (see appendix) these capillaries take on a brilliant red color and can be readily made out.

(2) Within the alveolar wall and in the peribronchial and perivascular lymphatics one can see small masses of pigment, at times free in the tissue and again contained within cells.

(3) Within the alveolar spaces the following structures can be seen. (a) A moderate number of red blood corpuscles some of which are undergoing disintegration. (b) Pigment granules which are derived from the disintegrating red cells. (c) A number of leukocytes and epithelial cells shed from the lining membrane of the alveoli, both of which in many cases contain pigment granules.

FIGURE 13.

Chronic Passive Congestion.

A. Engorged Capillaries in alveolar walls.
B. Pigmentary Cells.
C. Red Corpuscles.
D. Exfoliated Epithelial cells

(4) Frequently it will be seen that there is an increase in the connective tissue elements in the alveolar walls. Occasionally fibrin, such as is found in the exudate of red hepatization is present in a few alveoli, but this is exceedingly rare. At times also there will be found small hemorrhages filling up a few alveoli.

The bronchial tubes are found to be congested, the mucous membrane having a deep turgid appearance. The epithelial lining is usually wanting and the basement membrane is thickened and oedematous. The capillary vessels are all intensely engorged.

# HAEMORRHAGIC INFARCTION OF THE LUNGS

## PULMONARY APOPLEXY.

DEFINITION:- The term haemorrhagic infarction is applied to an effusion of blood into the pulmonary alveoli and interstitial tissue following embolism of terminal branches of the pulmonary artery. Actual haemorrhage as from rupture of an artery does not take place, but the blood escapes from the vessels by diapedesis. (See Lecture Notes on Thrombosis and Embolism).

PATHOLOGICAL ANATOMY:- Haemorrhagic infarctions are firm red, wedge-shaped masses usually situated just below the pleura with the base toward the surface of the lung. When recent they are brownish red in color and where they reach the surface of the lung the pleura covering them is generally inflamed. Occasionally infarctions occur singly, but more frequently they are multiple and are found scattered over the greater portions of a lobe.

NOTE:- In the case from which the present sections were taken there were multiple infarcts in all the lobes of both lungs dependent upon extensive pulmonary embolism.

The size infarcts vary from that of a pigeon's egg to that of an orange, or even much larger, (in the adult) The affected portion of the lung is impervious to air.

PATHOLOGICAL HISTOLOGY.- Under the microscope the alveoli are seen to be filled with red blood corpuscles. Sometimes, though not frequently, the alveolar septa are broken here and there. Blood is also seen in the alveolar walls which are compressed and in which fewer nuclei than normal can be made out on account of the coagulation necrosis (See Lecture Notes on Necrosis) which soon sets in after the infarct has been formed. If the infarct be very young no changes will be seen about its borders which are

sharply defined and surrounded by normal lung tissue. If however it has existed for some time the surrounding tissues will be oedematous and congested, or there will be evidence of inflammation on the accumulation of leucocytes which will be seen to form a definite zone about it. (See Lecture Notes on Thrombosis and Embolism). In cases of haemorrhage into the lung tissue from rupture of a small vessel the same changes will be seen.

ETIOLOGY:- Haemorrhagic infarction of the lung is most frequently associated with cardiac disease; it appears most often when the right cavities of the heart have become weakened and dilated, and thrombi have formed in the right auricle and ventricle. It is from these thrombi that emboli are carried away into the branches of the pulmonary artery. Thrombi may themselves also be formed in the pulmonary arteries, and it is possible that occasionally thrombosis of the peripheral veins may lead to embolism of the pulmonary arteries with the resultant infarcts.

A
3

# DUST PIGMENTATION.

In the lungs of all, especially of those who live in cities there is found a certain amount of pigmentation from dust. It is this which gives to the lungs the mottled appearance noted at autopsies and which in some instances gives to the entire organ a dark bluish black appearance. This is a condition falling short of the deep pigmentation caused by the inhalation of coal dust and yet it is of such constant occurrence that it deserves a passing word of description.

The pigment is derived from various sources and is chiefly composed of particles of soot and dust breathed in in the course of years spent in dusty dirty cities. While normally this amounts in years to only enough to cause a very distinct spotted pigmentation, in those who work in coal mines the lungs become after a year or so almost entirely black.

The distribution of the pigment is wide. It is found rarely in spots in the costal pleura, and very commonly in the visceral pleura, the deposit being in the deeper layer while the superficial layer is free. The bronchial glands contain large quantities of the pigment and are sometimes jet black. Along the course of the interlobular septa there is some pigmentation and in the perivascular and peribronchial lymphatics there is usually a considerable quantity. In those who have worked on coal mines the pigment is even more widely distributed, occuring sparsely in the alveolar walls, in desiquimated epithelial cells and in the lymph-adenoid bodies of the lung.

The inhaled pigment rapidly finds its way into the lymph channels and it is owing to this fact that we find the distribution just described. The particles are taken up, after passing between the cells of the alveolar membrane,

by the plasma spaces in the alveolar walls and ultimately find their way into the lymphatics which lie about the branches of the pulmonary artery and about the bronchi, which soon come to be injected with them. These lymphatic channels freely anastomose with the lymph spaces in the deeper layer of the pleura, which in their turn anastomose very sparingly with those of the superficial layer. This accounts for the fact this layer contains little or no pigment. All the lymph channels sooner or later find their way to the bronchial glands, and these as it were strain out the pigment, so that they soon become densely filled with it, and on section are seen to be black and not infrequently hard cutting with difficulty and with a gritty feel and sound. The ordinary inhalation of dust and the inhalation of large quantities of coal dust does not excite interstitial changes such as are set up by breathing particles of stone or flint.

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## ACUTE PNEUMONIA.

SYNONYMS:- "Common," "Lobar," "Croupous" or "Fibrinous" Pneumonia, "Pneumonitis," "Lung Fever," and "Pleuro-pneumonia."

Lobar designates the anatomical seat of the disease; croupous or fibrinous indicates the character of the exudate which accompanies the inflammation; (See Lecture Notes on Inflammations) while the prefix "pleuro" in the term pleuro-pneumonia, emphasizes the fact that the pleural covering of the inflamed lobe is frequently involved. Lung fever and pleuro-pneumonia are terms now most frequently applied to pneumonia in cattle.

DEFINITION:- Acute pneumonia is an infectious disease characterized by inflammation of the lungs and constitutional disturbances of varying intensity. (Osler).

ETIOLOGY:- The diplococcus pneumoniae, or pneumococcus discovered by Fraenkle, and by him asserted to be the cause of this disease, is found to be associated with every case and is now believed to bear a definite causative relation to it. It undoubtedly is the exciting cause, but it appears that predisposing causes are likewise necessary, as the organism has been found to be present in the mouths of at least 20% of healthy people. Such predisposing causes are most frequently found in exposure to cold, hardship, alcoholism, and not infrequently in traumatism. In addition certain diseases seem to render the system more susceptible to pneumonia. Of these Bright's disease, diabetes, prolonged attacks of fever, chronic nervous diseases and a previous attack are the most important. All of these conditions by lowering the vitality of the individual give to the specific cause of the disease, the pneumococcus, the conditions necessary for its rapid growth and development, with its consequent poisonous effect upon the system, shown most prominently as an inflammation of the lungs.

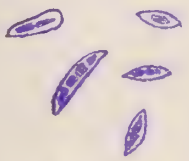


FIGURE 14.

Pneumococcus.
Showing Capsule, as seen in the blood of mouse inoculated with pneumococcus.

Males are more frequently affected than females, and the disease, which occasionally occurs in epidemic form, is found in all climates from the warmest to the most severe.

MORBID ANATOMY:—The chief stress of the disease falls upon the lungs the changes in which are usually grouped into three stages as follows:-

I. Stage of engorgement
II. " " Red Hepitization
III. " " Grey Hepatization

## STAGE OF ENGORGMENT.

PATHOLOGICAL ANATOMY:- On opening the chest the vessels of the pleura are seen to be greatly congested and

beneath these lies the deep red color of the lung tissue. To the touch the organ is firmer than normal, though portions not airless will still float in water. On section of a lobe there is an escape of bloody fluid, much of which is the fluid of oedema, the color being more of an arterial than of a venous red. Air still enters the lung and crepitation, though somewhat deadened can still be obtained.

PATHOLOGICAL HISTOLOGY.- Under the microscope the chief change noted is a marked engorgement of the capillary vessels which in places can be seen as tortuous red lines running in the alveolar walls and here and there protruding as small loops into the alveolar spaces. Occasionally one of these over-distended vessels ruptures and a small haemorrhage fills a few alveoli. The epithelial cells lining the alveoli are found to be swollen and in places are detached while within the greater number of air spaces lie also a few red and white corpuscles which have escaped from the capillary vessels.

This first stage of engorgement, or active hyperaemia, of the lung is soon succeeded by the second stage, that of

## RED HEPITIZATION.

PATHOLOGICAL ANATOMY:- Up to this time no inflammatory exudate has been formed but with the beginning of its formation we have the beginning of the stage of red hepitization. The lobe or lobes which have previously been in a state of engorgement now present certain very characteristic appearances. They are still more voluminous than normal, but do not collapse when the chest cavity is opened have entirely lost the soft feel of normal lung and become as firm and solid as liver tissue. No air can enter the alveoli and there is in consequence no crepitation. The surface of the affected lobe is frequently marked with the imprint of the ribs and the pleura, which was previously congested, is now distinctly inflamed and covered with

A = Fibrin.
B = Red corpuscles.
C = White corpuscles
D = Circulus sulcatus

thin film of fibrin. The color of the lung is somewhat darker than in the previous stage and a marked change in its physical condition is shown by its greatly increased friability. (it is more easily torn).

The cut section of the lung is dryer than in the previous stage and of a deep reddish-brown color. To the feel and when viewed with the light falling obliquely upon it one can make out a distinct granular condition of the surface. This is due to the protrusion from the cut alveoli of little plugs of fibrinous exudate. The small bronchi are also often found to be plugged with this fibrinous material. The lung cuts easily the knife not sinking into the tissue as when normal lung is cut. From the surface a bloody viscous fluid can be scraped, which on examination will be found to contain small granular masses. Portions of the lung tissue in this stage sink readily in water.

PATHOLOGICAL HISTOLOGY :- When studied with the microscope the alveoli are found to be everywhere filled with a fine net-work of fibrin in the meshes of which there are enormous numbers of red corpuscles, a few leucocytes and cells exfoliated from the alveolar membrane. The capillaries have lost the engorged appearance which they presented in the first stage, even appearing to contain less blood than normal. The alveolar walls are infiltrated with leucocytes. The pleura is thickened, the vessels in its deeper layer being engorged while on the surface there is frequently a layer of granular looking fibrin. (See Pleuritis)

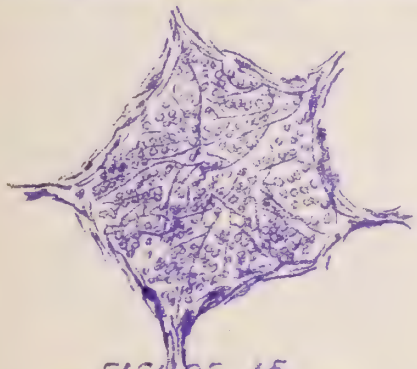


FIGURE 15.
Red Hepatization.
Showing an alveolus filled with red corpuscles entangled in a fine mesh work of fibrin.

When sections are stained by Weigerts fibrin method (see appendix on methods) the fibrin alone is stained and one gets a clear idea of the extent to which the alveoli and

filled with this material. On properly staining for micro organisms (see appendix) the presence of the pneumococcus can be demonstrated in the tissue and in the exudate. Not infrequently other organisms, such as the streptococcus and the staphylococcus can also be found.

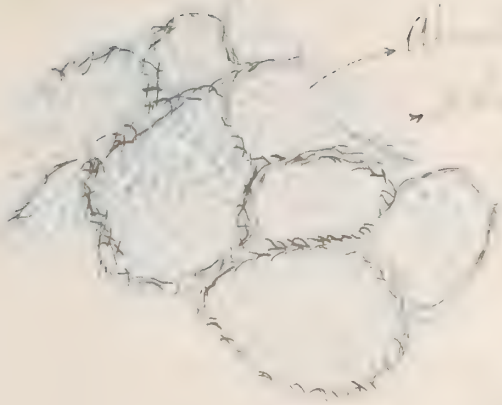
This stage

## GREY HEPITIZATON.

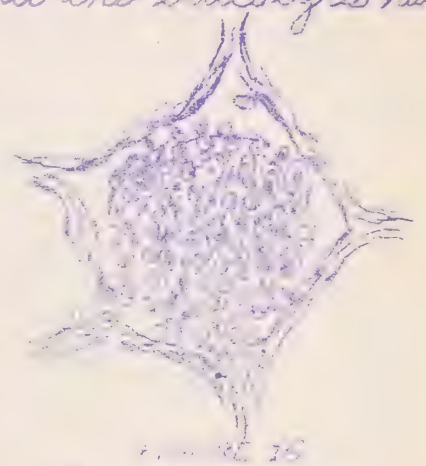
This stage of red hepitization passes gradually into the third stage of the disease, that of grey hepitization. The change does not occur all at once so that upon examining the lung we find that in portions the tissue is in the second stage, in others it is in the third stage while in other parts the transition from red to grey hepitization is in progress.

PATHOLOGICAL ANATOMY:- On inspection the lung is still found to be larger than normal; it is firm and solid and cuts easily as in the former stage; portions of the tissue sink readily in water and the cut surface has the granular feel and look already referred to. The pleura is coated as in the stage of red hepitization, with a film of fibrin, and in places the parietal and visceral pleuras may be found to be adherent. The color of the lung tissue has however changed from a dark reddish-brown to a greyish-white and is more moist than before. On scraping the surface with the edge of the knife one removes a creamy, greyish-yellow fluid. The tissue is even more friable than in the stage of red hepitization.

PATHOLOGICAL HISTOLOGY:- Although, when studied with the low power of the microscope, we see that the alveoli are still filled with a net work of fibrin filaments, which on using Weigert's stain are especially well differentiated, we find in the meshes of this material not the enormous numbers of red corpuscles as in the former stage, but almost as large a number of leucocytes with a few



exfoliated cells from the alveolar membrane. The red corpuscles have entirely disappeared and the leucocytes have taken their place. In the early part of this stage the exudate completely fills the alveoli as it did in the stage of red hepatization, but as the fibrin grows older it contracts somewhat and being attached to the alveolar wall at one or more points it is drawn away from the other portions leaving a space between it and the wall.



Alveolus partly filled with leucocytes and fibrin. Attached corner still present, others split in places

Grey hepatization may be looked upon as the beginning of resolution and when this proceeds normally the exudate undergoes disintegration and is expectorated in part and in part absorbed so that ultimately the lung is restored to its normal healthy condition. Occasionally however instead of proceeding on this normal manner the exudate becomes softer and is extensively infiltrated with a purulent fluid. This condition is spoken of as purulent infiltration and when it reaches such an extent that small abscesses are formed resolution rarely, if ever, occurs.

The terminations of pneumonia may be, as already indicated either;

1. Resolution which is the most common.
2. Purulent infiltration occurring in about 4% of all cases, or
3. Gangrene, which occurs in about 1% of all cases, or
4. Fibroid induration, which is the least common occurring in about 1% of all cases.

CHANGES IN OTHER ORGANS:- Although the lungs bear the brunt of the disease and show the most marked changes, other organs also are affected in many or

another, and the following changes are most frequently met with.

The liver is frequently enlarged and softer than normal.

The kidneys (see Kidneys) and are indefinite granular degeneration, especially of the cortical portion.

The heart is usually found to contains firm masses of fat and the right ventricle is frequently very much dilated, owing to the impediment that has existed to the flow of blood through the lungs and the consequent extra work which has been thrown on this part of the heart.

Pericarditis and more frequently Endocarditis are found to accompany pneumonia.

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# BRONCHO-PNEUMONIA.

SYNONYMS.- Catarrhal Pneumonia. Lobular Pneumonia.

The term lobular pneumonia indicates the anatomical distribution of the disease, individual lobules being affected and not a whole lobe as in the acute croupous variety. The term catarrhal is used because of the character of the exudate, which in typical cases contains no fibrin. (See Lectures notes on Inflammation.) Where the exudate is purely catarrhal or type the term Catarrhal Broncho-pneumonia would be applicable, because we frequently find pneumonia, lobular in distribution, the bronchi having been seriously inflamed, but in which the exudate, instead of being catarrhal, contains a greater or less amount of fibrin. To this condition we might with propriety apply the term Croupous Broncho-pneumonia

DEFINITION:- An inflammation involving individual lobules or groups of lobules and the terminal bronchi which supply them.

ETIOLOGY:- The disease although occurring at all ages is most common in the early and advanced years of life. At these extremes also it is most fatal, especially if accompanied by privation or exposure. In children it frequently follows an attack of one of the exanthematous fevers and according to some authorities the deaths from this sequel are more numerous than from the fevers themselves. Inhalation of irritant particles and of irritant gases frequently causes inflammation of the bronchi which rapidly extends to the pulmonary alveoli setting up an extensive broncho-pneumonia. In this way the inhalation of large numbers of tubercle bacilli gives rise to the most severe and fatal form of the disease. (See Tuberculosis) Finally broncho-pneumonia may be caused by foreign matter entering the trachea and ultimately being aspirated back into the smaller bronchi where it excites a most intense inflammation which soon involves the air vesicles. This is usually spoken of as "aspiration" or "deglutition" pneumonia and is met with in persons in whom, for any reason, the sensitiveness of the larynx is diminished.

PATHOLOGICAL ANATOMY:- Upon inspection at autopsy, the lungs are usually seen to be somewhat increased in size. Pleurisy and pleuritic adhesions are rare, though here and there where the surface of the lung shows slightly elevated patches the pleural covering is bluish and turbid in appearance. The organ except in cases of very extensive involvement feels vesicular throughout, generally even more so than normal because of the difficulty with which the air escapes from the tissue. Throughout the substance of the organ firm solid masses can be felt, which represent the patches of broncho-pneumonic consolidation. When one of these patches is at the surface of the organ it is represented by one of the elevated patches above mentioned. These patches

which are more elevated in the center than about the margins are frequently seen to be surrounded by a zone of emphysematous tissue.

On section the surface is of a dark red color and blood escapes freely. Projecting above the general level of the section are the lighter reddish areas which are the patches of bronchopneumonia. Between these the lung tissue is usually normal or slightly emphysematous, though at times the patches may be so numerous as to occupy the greater portion of an entire lobe. If the knife has cut one of these patches so as to show the bronchus one can make out the following changes.

(1) The bronchioli is somewhat dilated and filled with a mass of exudate which can be squeezed out as a muco-purulent yellowish fluid.

(2) Surrounding this there is an area of grayish-red consolidation which is usually elevated somewhat as already mentioned. Like the consolidation of acute croupous pneumonia this is sometimes granular to the touch and when looked at with the light striking it obliquely, and for the same reason. (What is the reason?) Frequently however the surface of this consolidation is perfectly smooth. (Why?) If the disease is advanced one may see here and there small white points from which purulent drops escape when the tissue is compressed between the fingers.

(3) Surrounding these areas of consolidation the tissue is darker red in color, smooth, airless and not so prominently elevated. This represents an earlier stage of the disease, and it is probable that this consolidation, or hepatization as it is called is always preceded by this reddened, collapsed condition which is sometimes spoken of as splenization.

The anterior upper borders, and the surface of the lobe are usually emphysematous, while at times one will find small haemorrhages in the pleura and in the lobules adjacent to those which are inflamed.

PATHOLOGICAL HISTOLOGY. - In a cross-section of a small bronchio-pneumonic patch we will see the following under the microscope.

(1). About the center if the section passes through it a bronchiole usually dilated and plugged with exudate. Its walls swollen and infiltrated with numerous leucocytes.

(2). In the alveoli surrounding this bronchiole a mass of exudate the character of which is described below, while the alveolar walls present very much the same swollen and infiltrated appearance as does the wall of the bronchi. These signs of inflammation of the walls of the alveoli and of the bronchial tubes (infiltration with small round cells, and engorgement of the vessels) is a point to which Delafield called attention as marking a distinction between this form of pulmonary inflammation, and acute croupous pneumonia.

CHARACTER OF THE CATARRHAL EXUDATE. - In order to thoroughly understand the character of the exudate in pure catarrhal pneumonia one must be familiar with the normal histology of the lining membrane of the pulmonary alveoli. These spaces are covered by a layer of flat pavement cells which wind around the partitions between the air cells and flatten themselves into all the irregularities of the surface of the alveolar walls. Besides these cells however there are others differing somewhat from them in structure and which are more numerous in the young than in the old. These are irregular in shape, somewhat smaller and have more finely granular protoplasm than the cells just referred to, and are found either singly or in groups. They also

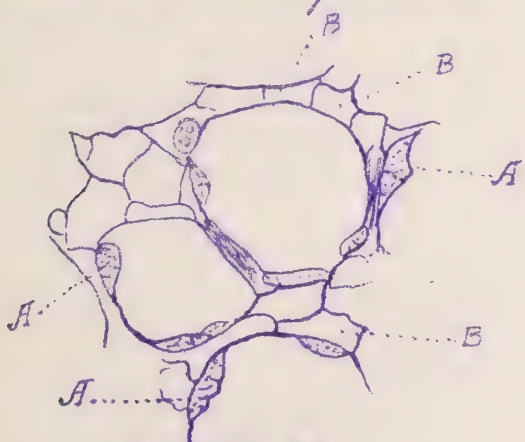



FIGURE 17.

Alveolar Cavities - Walls stained with Silver (x450) - (AFTER HAMILTON)

(A.) Young granular Epithelial Cells.
(B) Fully developed Squamous Epithelial Cells

take up staining reagents more readily and when viewed in profile project somewhat into the alveolar cavity. When large they sometimes show a nucleus, but this is frequently not seen. In all probability these bodies are young, growing epithelial cells, though various theories have been advanced to account for them.

Now when we carefully study the exudate of a pure catarrhal pneumonia with the high power lense we can make out the following structures which lie in a mucous substance:

(1.) Large cells with finely granular protoplasm in which can be seen one or more nuclei. Sometimes these nuclei show all the evidence of division, and the irregularity at times noted in the size of the nuclei within a cell would tend to confirm the opinion that nuclear and cell division was going on.

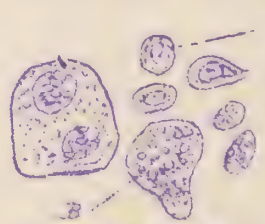



FIGURE 18.

Cells from the exudate of Acute Catarrhal Pneumonia.

A. Large cell with granular nuclei to which has since become fatty.
C. Smaller cells with single nuclei some are fatty.

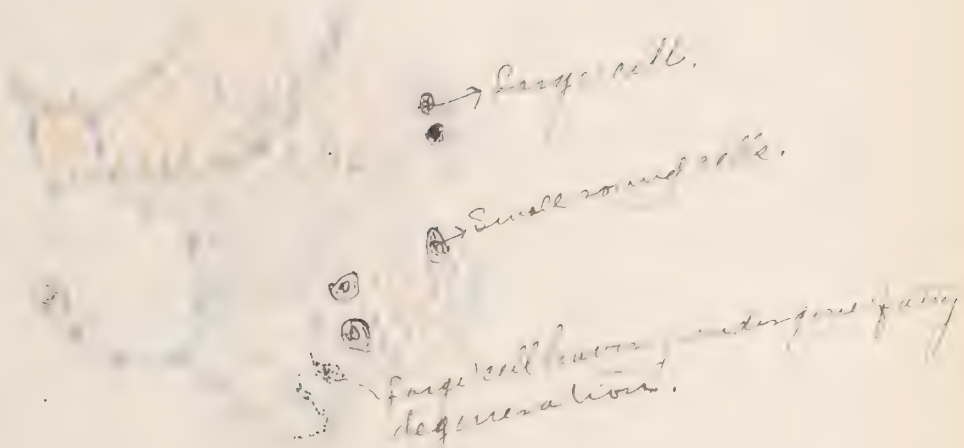
(2.) Large cells very much like those just mentioned, but instead of showing the signs of active growth showing those of degeneration. These frequently contain fat droplets and in some cases can be seen to be disintegrating. These are probably the same as those first described.

(3.) Much smaller cells, very finely granular, which like the first frequently show signs of increasing in number.

(4.) The same cell only showing signs of fatty degeneration.

(5.) Finally a certain number of leucocytes are seen, but these are usually fewer in number than the other cells mentioned.

The great mass of cells then are evidently not derived from the blood, but are more epithelial in type. These are undoubtedly derived from these germinal cells above described as occurring singly or in groups, and as smaller and more irregular in shape than the pavement cells which form the


Large cell.
Small round cells.
Large cells with irregular pigmentation.

greater mass of living cells. When a catarrhal pneumonia is set up these cells rapidly increase in number and by repeated sub-division give rise, with the cast-off pavement cells and the leukocytes derived from the blood to the great mass of cells which form the pneumonic exudate. Occasionally when a good profile view is obtained of an alveolar wall, these germinating cells can be seen as swollen granular bodies clinging to the alveolar wall, while near them are frequently to be found small clumps of cells evidently derived from them and lying free within the alveolar space. Some alveoli will be found which are entirely lined by these cells and when this is the case the older flat cells have been cast off and are to be found as fatty degenerating bodies in the exudate.

TERMINATIONS:- These may be as follows.

(1.) Resolution which occurs in the great majority of cases.
(2.) Abcess formation which is comparatively rare.
(3.) Gangrene which is also infrequent.
(4.) Interstitial pneumonia, (Chronic) which is the most infrequent of all terminations.

Caseation does not occur unless the broncho-pneumonia be of tubercular origin, or unless a simple broncho-pneumonia becomes secondarily infected with the tubercle bacilli. (See Tuberculosis).

# CHRONIC INTERSTITIAL PNEUMONIA.

SYNONYMS:- Cirrhosis of the lung. When of tubercular origin the term Fibroid Phthisis is applied to this condition.

DEFINITION:- A morbid condition characterized by a gradual increase in the connective tissue of the lung, leading to induration and a slow obliteration of the pulmonary alveoli.

VARIETIES:- Depending upon various circumstances, there are a number of different forms which fibroid changes may take. For our purpose however it will be enough to recognise two varieties, 1. the local, which involves but a limited portion of the lung, and 2. the general or diffuse, which includes either an entire lobe, or at times the greater part of the whole organ.

ETIOLOGY: Chronic interstitial pneumonia is very rarely if ever a primary, independent affection. It is dependent upon one or another of the following conditions.

The local variety is always an accompaniment of tuberculosis, and it is also found in connection with such conditions as syphilitic gummata, tumors, abscess cavities, hydatids, old hemorrhagic infarcts, etc.

The diffuse variety occurs under the following conditions:
1. As a sequence of acute croupous pneumonia. (rare).
2. Dependent upon inflammation of the pleura. This form is usually spoken of as Pleurogenous Interstitial Pneumonia.

The invasion takes place from the pleura, bands of fibrous tissue growing down into the lung from the pleura and spreading out into the pulmonary tubule, frequently following the course of the interlobular septa, obliterating as it grows the alveolar spaces.

3. Following extensive inhalation of irritant dust such as flint dust, the dust from needle pointing, etc., grinding, etc. To this form of the disease the term "Pneumonokoniosis"

has been applied. It is essentially a chronic broncho-pneumonia leading to fibroid induration, and is at first practically local, ultimately however involving extensive areas of lung tissue.

The term Chronic Interstitial Pneumonia should be applied only to such cases as are non-tubercular in origin, the term Fibroid Phthisis being reserved for such cases as supervene upon an initial tuberculosis.

PATHOLOGICAL ANATOMY:- Except in cases of fraemonokomiasis the disease is usually unilateral, the chest on the affected side being retracted. At autopsy the diseased lung is found to be shrunken and is frequently quite small, while the opposite lobes are large and emphysematous, usually covering a portion of the mediastinum. The heart is drawn outward toward the chirrotic lung. The pleural membranes may be extensively adherent, in which case the interstitial changes are probably of the pleurogenous variety. In other cases there is little or no pleuritic adhesion or thickening. The affected portions of the lung are airless and the lobe is small, firm and not infrequently quite hard. It cuts with difficulty and the cut section has a grayish semi-translucent homogeneous appearance is generally pigmented and shows here and there the cut ends of dilated bronchi. Very rarely small cavities which are not tubercular in character, may be found. (Charcot. Osler.)

PATHOLOGICAL HISTOLOGY:- If the chronic interstitial changes follow an initial acute pneumonia the fibrous tissue is developed "when the cellular infiltration of the parenchyma of the lung is long continued, the circulation at the same time remaining ample for the nutrition of the part." Under these circumstances large fibroplastic cells are first met in the alveolar walls and in the connective tissue surrounding the bronchi, the blood and lymphatic vessels, and in the interlobular septa. These cells develope into connective tissue in the usual manner (See Lecture Notes on the Development of Connective Tissue) and in this way gives rise

to a thickening of the affected parts and to a consequent diminution of the respiratory space. Frequently where the thickening of the alveolar walls is marked it will be seen that the alveoli are lined by short cylindrical or cuboidal epithelial cells instead of by the flat pavement cells always found in the healthy lung. Where this change is marked the alveoli will strongly resemble the acini of glands. In other portions of the section the alveolar spaces will have been completely obliterated by the new-formed connective tissue. Occasionally fibrous tissue is seen to develope, not only in the walls of the alveoli and in the other localities mentioned, but also in the exudate which still occupies a portion of the air vesicles. Or again one can make out that bud-like processes of connective tissue leave the alveolar walls and make their way into the exudate in various directions, while at the same time there is a formation of new capillaries from those in the alveolar walls, which are to supply the new tissue with nourishment. In this way also the alveolar spaces are obliterated and substituted by connective tissue.

Where the interstitial changes follow pleuritic inflammation definite bands of fibrous tissue will be seen penetrating the pulmonary tissue from the pleura. These correspond to the interlobular septa and form a part of coarse mesh-work in the interstices of which the lung tissue becomes compressed, sometimes to such an extent that it collapses and remains functionless. The same changes noted above (the substitution of cylindrical, or cuboidal cells for the normal pavement epithelium) can be seen in those alveoli which remain open.

The bronchi as a rule will be seen to be distorted and in some cases dilated on account of the traction exerted by the shrinking connective tissue as it grows older

# PULMONARY TUBERCULOSIS.

SYNONYMS:- Phthisis Pulmonalis. Consumption.

DEFINITION:- An inflammation of the lungs due to a specific micro-organism, the tubercle bacillus, and characterised by the presence of nodules of inflammatory exudate which undergo degeneration into cheesy masses

ETIOLOGY:- The immediate exciting cause of this disease is the tubercle bacillus, first discovered by Koch in 1882. In order however for the bacillus to exert its characteristic influence the individual must be either predisposed to the disease on account of some hereditary peculiarity in the resisting power of the pulmonary tissue, or must have his normal resistance reduced in some way, exactly how in every case not being clear.

VARIETIES:- Tuberculosis affects the lungs in several quite distinct ways, the difference being due to the channel by which the tubercle bacilli enter the organ and by the amount of resistance offered to its ravages. We can thus recognise the following varieties, the pathology of which we will consider separately. These three are

I Acute Miliary Tuberculosis
II Tubercular Pneumonia and Broncho-Pneumonia
III Fibroid Phthisis.

Before describing the difference between these forms of tuberculosis of the lungs it will be well to understand the pathology of a typical tubercle.

PATHOLOGICAL HISTOLOGY:- Mode of formation of tubercle

The bacilli having once gained access to the pulmonary tissue the following set of changes is set up:-

(1.) The fixed cells of the part, the connective tissue cells and the endothelial cells of the vessels, begin to proliferate and produce new cells in large numbers. These new cells are round or cuboidal in shape and have each a vesicular nucleus

that is a nucleus which does not stain solidly as does that of the small mono-nuclear leucocytes, but which has a definitely stained border containing a net-work of fine fibers among which can usually be seen one or more nucleoli. These cells are called epithelioid cells because of their resemblance to epithelial cells and not infrequently contain the bacilli.

(2.) Owing also to the irritation, the inflammation, set up by the bacilli, there is a migration of leucocytes from the vessels to the area of infection. These cells are called the lymphoid cells.

(3.) By the fibrillation of the connective tissue a sort of reticular net work is formed in which these elements lie. This can be seen very distinctly in some cases while in others it is made out with difficulty, or only seen at the margins of the tubercle

(4.) Finally in some tubercles, but not in all, and consequently not an essential part of a tubercle, one sees especially large cells containing many nuclei, either grouped in the center, or arranged about the margins. These are known as the giant cells and are probably produced by the increase in the protoplasm of the cell and the repeated sub-division of its nucleus, without cell division being completed. In many cases these cells contain the tubercle bacilli. It would seem that the more intense the tubercular poison the fewer the number of giant cells formed.

In a typical tubercle then before it has undergone any retrogressive changes we have a nodule composed of epithelioid, lymphoid and sometimes giant cells usually lying in a loose reticulum of fibrous tissue.

When once formed tubercles undergo casiation and sclerosis. In the central part of a tubercle, owing to the action of the tubercle bacilli, and to the fact that the tubercle is non-vesicular the cells undergo a process of death known as coagulation necrosis (See Lecture Notes). As this advances the cells lose their outlines, their nuclei cease to stain and they are finally converted

into a structureless finely granular mass. This process gradually spreads peripherally until the whole tubercle is changed into a yellowish homogeneous cheesy substance. This is spoken of as caseation. After this the tubercle, or the aggregation of cheesy tubercles usually seen may undergo softening, or may become encapsulated by fibrous tissue or may harden into calcareous masses. If as sometimes happens the tubercle does not become caseous there is a great increase in the connective tissue of the part which grows into and through the mass of cells finally converting it into a fibroid nodule. In nearly every case whether the tubercle becomes caseous or fibroid there is a certain amount of connective tissue developed about it. This is especially found in those cases where the inflammation set up is of a minor grade of severity and reactive in nature resulting in the production of cicatricial tissue which tends to restrict the advance of the tuberculous process. In other cases where the inflammation is more severe in character we frequently find in the tissue surrounding the tubercles in a limited area pneumonic exudation, either catarrhal or croupous in character depending upon the severity of the process.

So much then for the general histology of a tubercle. We will now consider the various pathological changes met with in the three varieties of tuberculosis of the lung mentioned above.

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## ACUTE MILIARY TUBERCULOSIS.

PATHOLOGICAL ANATOMY:- This condition is set up when tubercle bacilli enter the lungs through the circulation and become lodged pretty generally throughout the organ as small emboli masses in the pulmonary capillaries. The organ is usually somewhat more voluminous than normal, is generally congested, is firmer and contains less air than when healthy. The pleura is very frequently seen to be studded with

greyish nodules the size of milled seeds, which if recent are semi-translucent but if old of a distinct yellowish tint.

On section of the lung the surface is usually seen to be of a deep red color and profusely studded with very minute greyish points which stand out somewhat from the surrounding tissue and are each separated from its neighbor by somewhat emphysematous lung tissue. Occasionally these miliary tubercles are so small as to escape notice entirely until the organ is studied under the microscope. In some cases the deposit of tubercles will be very great, while in others it will be scanty. They are uniformly distributed throughout all the lobes and have as nearly as possible the same size everywhere. On closely examining the arrangement of these bodies it will be seen that they most frequently follow the course of the smaller branches of the pulmonary arteries. Less frequently they are aggregated in little clusters, but the individual tubercles do not unite to form larger nodules, as in other forms of the disease, but remain discrete with well defined margins

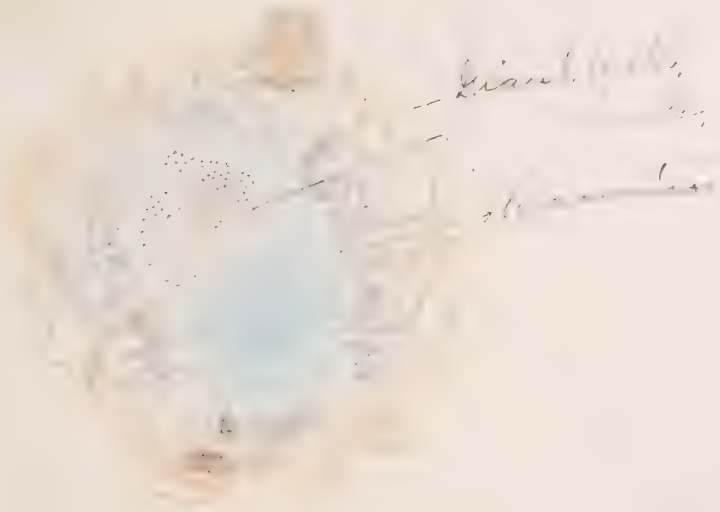
PATHOLOGICAL HISTOLOGY:- The structure of these tubercles has already been given. The tissue surrounding them is usually slightly emphysematous as in catarrhal pneumonia. There is also some congestion of the capillaries in the alveolar walls and occasionally there will be a slight grade of inflammation in the alveoli immediately surrounding a tubercle.

---

## TUBERCULAR PNEUMONIA.

We may divide this form of pulmonary tuberculosis into three stages as follows:

1. That of acute or sub-acute catarrh.
2. That of cavitation.
3. That of ulcerative excavation.



## First Stage, that of Acute or Sub-Acute Catarrh.

The first anatomical signs of the ~~form~~ of tuberculosis are practically those of an acute, or sub acute catarrhal pneumonia which has already been discussed (See back). The changes are in the great majority of cases first seen at one or the other apex. Whether this early stage of the disease is due to the action of the tubercle bacilli, or whether there is a simple catarrhal pneumonia upon which there is a tuberculous process engrafted has been a matter of dispute. Undoubtedly it is possible for an area of lung tissue suffering from catarrhal pneumonia to become infected with the tubercle bacilli and thereupon to take on the characteristics of a tuberculous process and to ultimately terminate in caseation and ulcerative change, but that all cases of tubercular pneumonia begin in this way is highly improbable as we know that the tubercle bacilli when they find lodgment in healthy lung tissue in some cases, probably due to predisposition, proceed to multiply and set up changes which can not be differentiated from those of ordinary catarrhal pneumonia. It would be best therefore to consider that in some cases there is a primary simple catarrhal, and that in others the process is tuberculous from the first.

After the primary catarrhal changes have lasted for a time other changes set in characteristic of all tuberculous processes, namely, the formation of tubercles and their degeneration and caseation.

Stage of Consolidation. – Pathological Histology. – At autopsy one usually finds the lung more less bound down by adhesions. These are sometimes confined to the apex but in the majority of cases are quite general. Over the more dependent parts of the lung there is frequently found a thin film of fibrin. When removed from the body the organ does not collapse as does the normal lung, but feels solid to the touch. In particular the lung of acute progressive pneumonia, it also resembles this condition in being heavier and firmer than normal. Crepitation can only be obtained here and there where the

intermediate areas one can feel firm solid masses. Inspection of the surface shows scattered everywhere numerous rounded nodules of hard consistency sharply defined from the surrounding lung tissue.

Upon section the first appearance to attract attention is that of these nodules scattered throughout the lung tissue. If the disease be in a comparative early stage these appear more as patches of a greyish color with indefinite outline, but when advanced they become more definite in appearance and form hard masses with sharply circumscribed borders. In size these may from that of a pin's head to that of an egg. At times they are found to have coalesced to a greater or less extent, so that nearly an entire lobe will have become solidified, producing a condition spoken of as caseous tubercular pneumonia. In other cases the caseous areas will present a lobular arrangement about a bronchus, the condition being one of tubercular broncho-pneumonia. As a rule the nodules are more numerous at the apex of the lobe and in the peripheral portions though in advanced cases they are to be found everywhere. The tissues lying between these masses is usually congested, frequently quite oedematous, and not infrequently the seat of a fibrinous inflammation, appearing solid, though of a different color from the cheesy tuberculous masses. Within some of the caseous areas bronchi can be seen, generally filled with necrotic material.

PATHOLOGICAL HISTOLOGY:- Microscopic examination of a lung in this condition reveals the general appearance of tubercles with which we have become familiar. It will be well however to direct our attention not so much to the caseous areas themselves as to their borders and to the tissue lying between them. In nearly every instance of this disease it will be noticed that about the borders of these masses there is an attempt at the formation of fibrous tissue, which in some instances may have resulted in the production of quite a zone of spindle cells lying in a concentric manner about the

tuberculous area. In practically every case this cirrhotic complication is present though it is in the more chronic cases that it reaches its greatest development. The walls of the bronchi and of the blood vessels may also be seen to be thickened by an increased growth of their connective tissue. Lying somewhat within the zone of fibrous tissue increase there will be found a more or less definite zone of small round cells and still further toward the centre of the tubercular area the cells will be found in a state of degeneration and fragmentation of their nuclei will be readily seen. Within the alveoli surrounding these diseased portions of the lung, the capillaries will be seen to be congested. Within some of the alveoli there will also be found a fibrinous exudate.

STAGE OF ULCERATIVE EXCAVATION.- (Chronic Ulcerative Phthisis). Sooner or later all tuberculous processes, except those which are very acute and rapidly terminate in death, proceed to caseation and softening. The great majority of cases thus become chronic in character and are associated with cavity formation and chronic interstitial changes of greater or less extent. Although these cases are primarily of tubercular origin there is usually a secondary infection, as the disease advances with the streptococcus, or the staphylococcus, or with both, to which infection many of the symptoms noticed in these cases are due.

When one examines a lung in this stage of the disease all the appearances noted in the stage of caseation are found to be present and to be accentuated. One finds the large areas of caseation, with beginning softening about their centres, the areas of tubercular broncho-pneumonic consolidation and caseation in the same condition; the very large number of small tubercles scattered everywhere throughout the lung, a second crop so to speak, representing a process of extension from the older foci; congestion, oedema, fibrinous exudation into the alveoli surrounding tuberculous areas, and very marked cirrhotic changes everywhere

The most marked change however, and the one from which this stage of the disease gets its name, is that of cavity formation produced by the death and ulceration of the pulmonary tissue. These cavities usually begin at one or the other apex, and by the extension of the necrotic process often increase to such a size that they occupy the most of an entire lobe. These extensive cavities in most cases consist of a series of communicating excavations with ragged walls, (in which there are numerous tubercles in all stages of degeneration, and by whose breaking down the cavity is enlarged) and are filled partly with a muco-purulent material that is sometimes bloody. The process of ulceration usually begins in a bronchus which has become filled with caseous material, and in whose walls there is going on an active tuberculous process. In other cases the excavation begins in the centre of a caseous mass and not in a bronchus. Running across these old cavities one may frequently see bands, the remains of bronchi, or of blood vessels which have not been disorganized by the necrotic process. It is by rupture of one of these isolated vessels that pulmonary haemorrhage is frequently caused. Oozing from the smaller ones and from the walls of the cavity makes the contents of these spaces very frequently bloody. Rarely one meets with another variety of cavity which is usually situated at the apex of the lobe and is surrounded by dense connective tissue. The walls of these cavities are smooth. In some instances they communicate directly with bronchi. They are old excavations which have become quiescent though they never heal entirely.

The pleura in nearly all cases of chronic tuberculosis is involved. There may have been only a slight inflammation resulting in the formation of weak thin adhesions, or the process may have been more severe and the resultant adhesions dense and firm so that at times the pleura becomes thickened from a mere delicate membrane to a structure several millimeters in thickness. The pleuritic inflammation may either be simple in its nature, or tubercular in which latter case there will be

found in the pleura either collections of miliary tubercles or small caseous masses. There may be adhesions of fusion.

## FIBROID PHTHISIS

The changes in fibroid phthisis are so similar to those in chronic interstitial pneumonia that they will not be described separately. Fibroid phthisis is essentially a chronic interstitial pneumonia following upon a primary tuberculous inflammation of the lungs, just as ordinary chronic interstitial pneumonia is frequently the sequel of ordinary acute croupous pneumonia. The term Fibroid phthisis had better be retained to designate the chronic fibroid induration which is of tuberculous origin while the term Chronic Interstitial Pneumonia should be used to designate like changes resulting from all other causes. —

(See Chr. I-t. Pneumonia)

# ACUTE PLEURITIS.

SYNONYMS:- Pleurisy. Inflammation of the Pleura

DEFINITION:- An acute inflammation of the parietal surfaces of the lungs and pleural cavities which is usually associated with a fibrinous, sero-fibrinous or purulent exudate.

ETIOLOGY:- The fibrinous variety may be due to exposure to cold, or may be associated with diseases of the lungs such as acute pneumonia, cancer, pneumonia or tuberculosis. The sero-fibrinous may possibly be caused by exposure but it is more probable that micro-organisms play an important part in their causation and especially is tuberculosis

an important ætiological factor. The purulent variety may follow upon an acute sero-fibrinous pleurisy, it may occur during an attack of one of the specific fevers especially scarlet fever and typhoid fever. It may accompany pneumonia or frequently be due to rheumatism as any injury to the pleura by a fractured rib.

PATHOLOGICAL ANATOMY:- We may describe this in three stages. In the first stage on inspection the pleura is reddened somewhat, there is distinct engorgement of the vessels some of the larger ones being especially prominent to the naked eye and the normal glistening appearance of the pleura is replaced by a dull semi-opaque appearance and is dryer than normal, and granular. In the second stage these changes are still more marked and there has been a transudation of plasma from the engorged vessels and over the surface of the pleura there has formed a thin layer of fibrin which can be easily scraped off with the finger nail, disclosing a much congested, granular-looking membrane beneath. On section of the pleura in this stage it is seen to be thickened and somewhat oedematous and the lung tissue immediately beneath it is slightly congested and firmer than normal. In the third stage there has been an absorption of the serous exudate and the two pleural surfaces have come together and have become more or less adherent by fine bands of fibrin running from one to the other. Later in this stage however these fibrinous adhesions give place to stronger connective tissue adhesions which have been formed by the organization which has taken place in the fibrin lymph which at first held the surfaces in apposition. These adhesions are at times so strong that they firmly bind the lung to the parietal surface and when attempting to remove the organ from the chest cavity it is frequently torn if great care is not taken to break all the adhesions. When a lung in this condition is removed from the body it is seen to be covered by a very much thickened pleura from all parts of

which fibrous bands lead away. These were the bands which bound the lung to the parietal pleura. On section the pleura is seen to be very much thickened and in many places there are distinct bands of connective tissue running from the pleura into the underlying pulmonary tissue, following generally the lines of the interlobular septa and giving rise to that form of interstitial pneumonia called fibrinogenous interstitial pneumonia.

PATHOLOGICAL HISTOLOGY:- Beneath the microscope, in the first stage it is seen that there is considerable engorgement of the small vessels of the pleura with red blood corpuscles and at the same time it is noted that there is also a greater number of leucocytes present than in normal blood. Surrounding many of the engorged vessels can be seen accumulations of these leucocytes which have evidently escaped from the vessel. There is also seen to be a rapid proliferation of the connective tissue cells, denoted by the presence of large numbers of small round cells in the tissue which are not leucocytes, and finally the lymph spaces in the pleura seem to be increased in size and to contain more fluid than normal.

In the second stage these same appearances are still visible only more marked, and in addition the pleura is thicker than before, and on its surface there is seen to be a layer of fine fibrin filaments among which lie a greater or less number of leucocytes. Sometimes between this layer of fibrin and the endothelial cells which line the pleura there will be seen to be an accumulation of oblong, or spindle shaped cells, which are cells exfoliated from the endothelial layer just mentioned. In the third stage, that of organization, there is still seen on the surface a layer of fibrin, of greater or less thickness. Below the pleura in the pulmonary tissue there is evidence of congestion. The pleura is seen to be very much thickened and the distinction into two separate layers is less definite than normally, or than

in the earlier stages of the inflammation. The deeper layer of the pleura seems to be swollen, its individual fibers of connective tissue to be increased in size, between which the blood vessels are numerous and engorged. The superficial layer seems to be made up of a more delicate reticular tissue containing large fusiform forms of which contains a few red blood corpuscles, and between these there are numerous small loop-like blood vessels shooting up from the vessels beneath. Occasionally one of these will be seen to have ruptured, causing us to see all hemorrhage. Towards the surface the tissue is made up entirely of a granular looking fibrin into which some of the loops seem to be pushing. Between the young vessels that are thus forming there are numerous young connective tissue cells to be seen, some round and small, others more elongated and still others distinctly spindle shaped.

Examine all these changes with the low power of the microscope first and then study them more carefully and in detail with the high power, making drawings of the various shaped cells and of some of the small loops of the blood vessels.

Briefly to summarize we may divide pleurisy into the following three stages which have been described.

1. A stage of congestion;
2. A stage of exudation; and
3. A stage of organization.

Pleurisy is an inflammation of a serous membrane and for further details read the section on inflammation of the peritoneum, under inflammation.

A

# Laboratory Manuel

of

# Pathological Anatomy.

## Third Part

## Diseases of the Kidney

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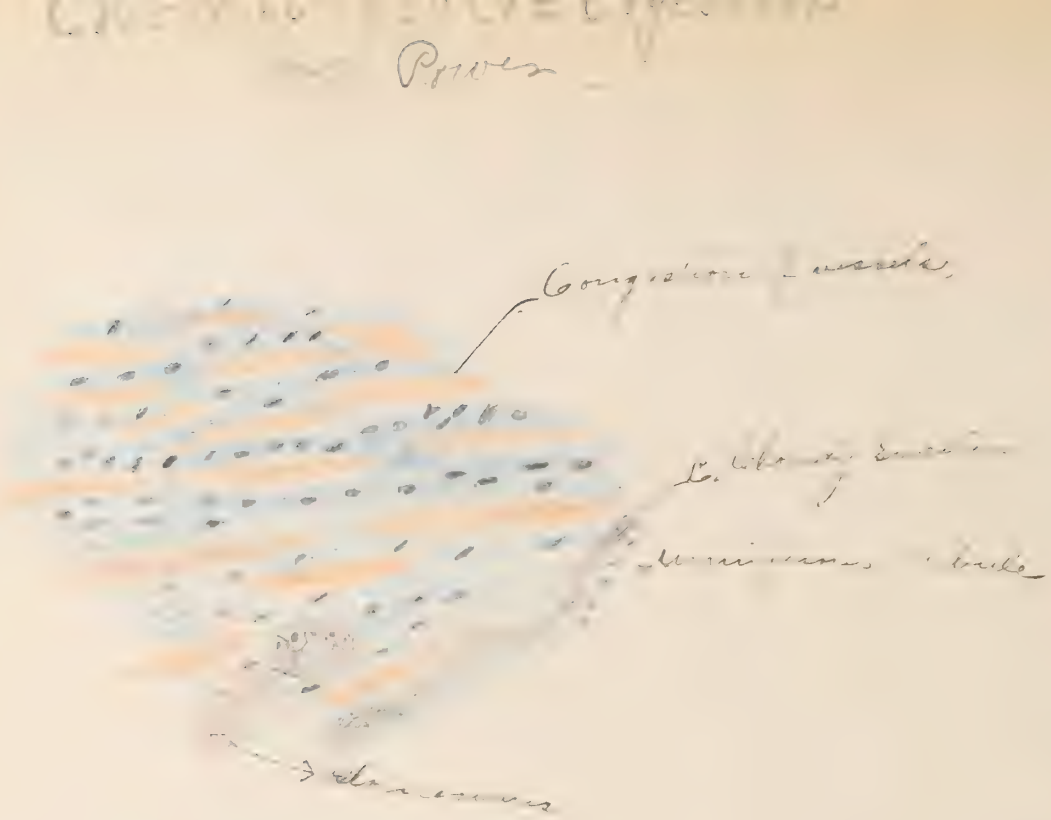


# The Kidney

For the normal anatomy and histology of the kidney read ~~work~~ on anatomy and histology

## Chronic Passive Congestion

DEFINITION — A condition of the kidney in which the vessels are engorged with venous blood due to some obstruction to the outflow of blood from the organ

ETIOLOGY — Any thing which obstructs the flow of the blood

PATHOLOGICAL APPEARANCE — The organ is increased in weight and may weigh as much as eight to ten ounces. In consistency it is harder and more elastic than normal. On the capsule of recent cases the capsule is removed from, and the cortex stripped off and is not thickened. In color the kidneys at first red or even purplish and the stellate veins in the capsule can be easily distinguished. Upon section the medulla and the cortex are both found to be broader than normal, though the latter is more affected than the former. All the vessels are found to be very full of blood and blood escapes freely from their cut ends. The malpighian bodies are large and more prominent than normal

PATHOLOGICAL CHANGES — The most prominent pathological appearance of the kidney in the congestion of all the vessels with the cortex and medulla. The capillaries are greatly distended with blood. In the cortex this is especially noted in the glomerules of the kidney. In some cases there may, sometimes be found, in addition to the congestion, a diapedesis of the red blood cells into the substance of the kidney, especially around the glomerules, and through the capsule. In the medulla the capillaries lying between the straight tubules appear as rather large tufts of red lines. With the congestion is of a severe grade there is found a more

be brought to bear upon the tubules surrounding the engorged capillaries that they become considerably compressed and the epithelial cells of the tubules, on account of this pressure are injured and in many places become atrophic and desquamated. In the convoluted tubules of the cortex the epithelial cells are frequently found to be swollen, but they are rarely shed from the basement membrane. Haemorrhages into the tubules and consequent blood casts are of course common. Blood pigment such as is found in chronic passive congestion of the liver and lungs is but rarely met with, owing probably to the fact that in the kidney the extravasated blood is very quickly washed away. The albuminuria associated with chronic passive congestion of the kidneys is to be explained by the obstruction which exists to the venous flow, and if the cause of the congestion be removed the kidney structure is so little altered that no serious results follow.

Chronic interstitial changes when found in connection with chronic passive congestion are not to be looked upon as the result of the congestion, but as having existed previous to the congestion or as having supervened during its course due to other causes.

# NEPHRITIS

DEFINITION - By this term is meant broadly speaking any inflammatory condition of the kidney. By many of the terms is used to cover all the conditions embraced by the term Brights Disease, while by still others it is employed to include other conditions as well. For our purposes we will give it the above definition.

As in the lungs and liver there were found to be various forms of inflammation, that is inflammations affecting the different structures of these organs and running different courses, so in the kidney we find that at one time the interstitial tissue seems to bear the stress of the inflammatory process, while at another it is the parenchyma, the cells of the tubules that are the most severely affected. We thus then recognise a parenchymatous and an interstitial inflammation of the kidney. For the sake of clearness we will consider the inflammatory conditions of the kidney under the following heads, and in the following order:

1. Inflammation affecting the parenchyma of the organ - parenchymatous inflammation. This is usually spoken of as Acute Parenchymatous, or Catarrhal Nephritis.
2. Chronic inflammation affecting the interstitial tissue and spoken of as Chronic Interstitial Nephritis.
3. The various forms of waxy infiltration of the kidney.
4. Inflammation affecting the various glomerular structures of which we recognise:
   (a) Acute catarrhal glomerulo-nephritis.
   (b) Acute interstitial glomerulo-nephritis.
   (c) Chronic interstitial glomerulo-nephritis.

# CATARRHAL NEPHRITIS.

SYNONYMS - Tubular Nephritis, "Parenchymatous Nephritis" Desquamative Nephritis. The Kidney of Acute Bright's Disease.

DEFINITION:- An acute inflammatory process the chief and primary stress of which falls upon the tubular epithelium.

ETIOLOGY - Exposure to hardships, cold and wet are the most frequent causes of this condition. Alcoholic excess and the administration of certain poisons also produce it. It is finally a frequent accompaniment of certain general diseases, especially the specific fevers, such as scarlet and typhoid and is sometimes found as a complication of jaundice.

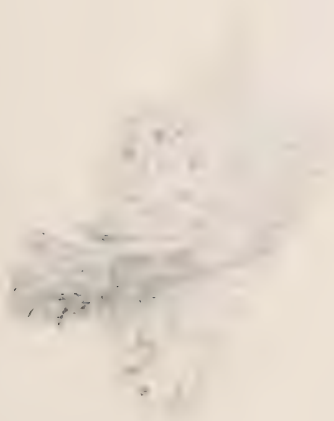
STAGES:- For convenience of description this disease is usually divided into three stages as follows:-

1. That of acute catarrh,
2. That of fatty degeneration of the tubular epithelium;
and 3. That of cirrhotic contraction.

## FIRST STAGE - THAT OF ACUTE CATARRH.

PATHOLOGICAL ANATOMY:- If the body comes to autopsy during this stage of the disease, which is rarely the case, the kidney is found to be increased in size and covered by a thin glistening capsule which is readily stripped off. The color of the organ is usually somewhat paler than normal, though this is not always the case. Upon section the cortex is found to be increased in thickness and to be of a dull greyish color, while beneath it the pyramids of the medulla appear in marked contrast because of the congestion of their vessels. Aside from these changes there are no distinctive gross appearances.

PATHOLOGICAL HISTOLOGY:- Beneath the microscope one sees that the convoluted tubules of the cortex are more prominent than normal because of the enlargement of their cells. These will be found to be much swollen, at times



Fodi - & tubulis absorbentibus
circumvicinis ope spirabilium
ex sto deglutitum.

their increased size completely occluding the lumen of the tube, and to be distinctly granular in appearance. This has led to the term of "cloudy swelling" for this condition and under this heading it is sometimes described as a separate condition from catarrhal nephritis though it is in reality only the very earliest stage of that disease.

FIGURE 19.

First Stage Catarrhal Nephritis.

Cloudy swelling of Tubercular Epithelium

The capillary vessels between the tubules and within the glomeruli are usually distended and sometimes ruptured. It is very rarely that one meets with a kidney in just this early stage of inflammation one usually sees a somewhat later stage where the cells which line the tubules have begun to be shed from their basement membrane and come to lie free in the lumen of the tube in some cases being so numerous as to completely occlude the same.

## SECOND STAGE - THAT OF FATTY DEGENERATION OF THE TUBULAR EPITHELIUM.

PATHOLOGICAL ANATOMY - If the disease is of longer standing say from 3-4 weeks duration we will find a somewhat different state of things. The kidney is still enlarged and is heavier than normal. In consistency it is flabby and the capsule as in the former stage strips off readily. The color of the organ is usually brownish or of a mottled yellow and brown appearance. Congestion of the smaller superficial vessels is also marked. When the organ is cut one finds that the cortex is

the size of the organ as a whole is due chiefly if not entirely to an increase in the thickness of the cortical substance, which is usually from one half to one again as thick as normal and is of a pale brownish yellow color. The medullary portion is little if any thickened and deeply congested.

PATHOLOGICAL HISTOLOGY:- The chief change to be noted when a section is studied under the microscope is the presence of droplets of fat within the cells of the convoluted tubules This fat is the result of process of fatty degeneration and the cells so affected are seen to stain poorly and in many instances to be broken up into a mass of fatty and granular detritis, which in places entirely blocks the lumen of tubule. Usually the tubules so affected are considerably dilated

FIGURE 20

Second Stage Catarrhal Nephritis

Epithelial cells falling away from basement membrane into lumen of tubule and then breaking up into fatty and granular detritus.

The malpighian bodies are enlarged somewhat chiefly owing to the tufts being swollen from oedema. No fatty changes however are noted on the glomerular epithelium.

In a certain proportion of cases, there is in addition to the tubular changes already mentioned an interstitial complication. Groups of small round cells can be seen chiefly accumulated in wedge shaped masses beneath the capsule, or in some cases scattered diffusely through out the connective tissue which lies between the tubules. These cells represent a proliferation of the fixed connective tissue cells of the organ, an inflammatory process involving the interstitial tissue as well as the tubular epithelium, a true combination of a catarrhal

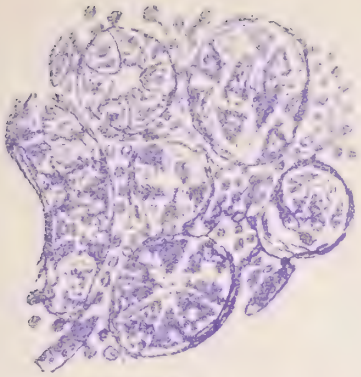


FIGURE 21.

Catarrhal Nephritis

Showing degeneration of tubular epithelium, and small cellular infiltration of the fibrous tissue between tubules.

and an interstitial nephritis, which clinically prove to be the most malignate and uncurable cases.

## Third Stage — That of Chronic Contraction

Many cases of catarrhal nephritis recover before reaching this third and last stage of the disease, but in those cases which linger for years, the changes instead of progressing to recovery become chronic in character bearing the symptoms of catarrhal nephritis and taking on the characteristic of a chronic interstitial inflammation. Those cases which become chronic are probably those which in the second stage began to show the interstitial complication just described. In some instances the catarrhal changes pass off entirely, or almost entirely and nothing remains but the diffuse increase in connective tissue which by contraction distorts the kidney and reduces its size.

A description of the changes seen in the contracted kidney will be given when we come to study the Granular Red Granular kidney of primary chronic interstitial nephritis.

---

THE URINE:- It is scanty in amount and in the earlier stages it frequently contains blood. Albumen is present in large amounts, and the specific gravity is ranging from 1025 to 1035. Fatty granular, granular and blood casts are found in considerable numbers.

DROPSY:- This is a marked feature of the disease and at times is very great. It frequently comes on suddenly and affects the loose tissues under the eyes, the tissues of the genital organs, and the lower extremities. Later in the disease it may become general.

PULSE AND HEART:- These are not materially affected in the earlier stages. When considerable contraction sets in the pulse tension is increased and there may be slight hypertrophy of the left ventricle.

## CHRONIC INTERSTITIAL NEPHRITIS.

SYNONYMS:- "Cirrhotic Kidney" "Gouty Kidney" "Small Red Granular Kidney."

DEFINITION:- A chronic inflammation of the kidney, the cause of which falls upon the interstitial tissue and which is marked by an increase in the same wherever it occurs in the organ.

ETIOLOGY- Lead is probably the strongest predisposing cause and it is claimed that the uric acid present in the body during this disease is the agent which irritates the connective tissue of the kidney and causes it to increase.

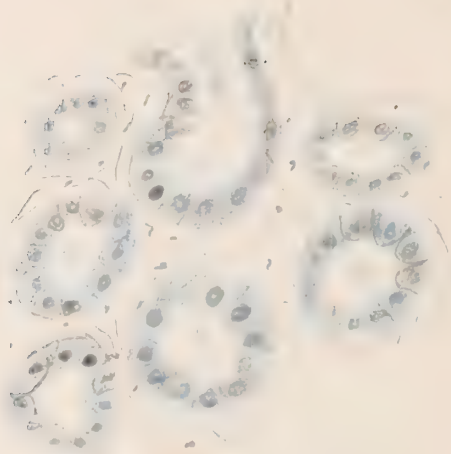
The excessive use of alcohol is instrumental in setting up this disease.

Certain poisons notably lead are known to be the direct cause of chronic inflammation of the connective tissue of the kidney.

And finally Syphilis seems also to cause this condition of the kidney in a certain number of cases.

STAGES.- As in the case of catarrhal nephritis we here also for convenience sake to divide the disease into stages, so here also we may recognize an early and an advanced stage of the disease.

PATHOLOGICAL ANATOMY- EARLY STAGE:- In the majority of cases the changes within the organ in the stage are so slight as to cause no noted evidence of the disease. In other cases the slight increase in the connective tissue leads to some enlargement of the kidney which with the slight adhesions which may exist between the capsule and the organ will



the only points of importance to be noticed at autopsy.

PATHOLOGICAL HISTOLOGY - EARLY STAGE - The changes to be noted with the microscope in this stage are also very slight. Here and there a few wedge shaped bands of fine fibrous tissue can be seen just beneath the capsule which run for a short distance within the cortex surrounding a few of the convoluted tubules the epithelium of which is generally swollen somewhat and slightly granular. The only other point worthy of note is a slight hypertrophy of the muscular coats of the arterioles.

PATHOLOGICAL ANATOMY - ADVANCED STAGE. - At autopsy the kidneys of advanced chronic interstitial nephritis are as a rule found to be very much diminished in size and to be more or less misshapen, tho' occasionally a case is met with where the kidneys are as large, or even larger than normal and increased in weight. This however is very exceptional. The capsule is found to be thickened and when one attempts to remove it it comes away with difficulty and frequently brings small portions of the kidney substance away with it. Upon removal of the capsule the surface of the organ is seen to be finely granular in appearance resembling somewhat the surface of the cirrhotic liver, on a much smaller scale. Here and there one usually finds small cysts containing a clear serous like fluid. Some of these are very minute while others are of considerable size and occupy a considerable portion of the thickness of the cortex. Upon section one is immediately struck with the thinness of the cortical substance; it is frequently reduced to less than half of its normal thickness. One may now note the small cysts within the cortex even better than before the kidney was cut. As consistency the organ is tough and inelastic and cuts with difficulty.

PATHOLOGICAL HISTOLOGY - ADVANCED STAGE. - The following minute changes are those most characteristic of this condition.

The capsule is seen to be very much thickened and in places to be depressed somewhat. These depressions resemble those seen in the surface of the cirrhotic liver. From the base of these depressions bands of fibrous tissue run into the substance of the organ where they form a more or less complete net work of connective tissue fibers in the meshes of which lie the much altered kidney structures, the tubules and glomeruli. Because of the pressure exerted by the contraction of these strands of connective tissue the tubules in many places are mere shrunken remains from the walls of which the epithelial cells, much atrophied and very granular, have separated. Large areas of cortical substance are in this way destroyed. The malpighian bodies likewise suffer from the pressure exerted upon them by the new tissue. The capsule which surrounds them shares in process of connective tissue increase and in many instances can be found to be very much thickened and to have encroached upon the glomerular tuft to such an extent that it is reduced to a fraction of its original size. In some places one can find places where this encroachment of connective tissue of the capsule has been so extreme that no trace of the glomerular tuft remains, its place being occupied by a concentric mass of fibrous tissue.

Within the medulla also the increase about the tubules is especially noted, and as in the cortex there will be found areas of various size in which there is little if any kidney tissue left. Like cicatricial tissue in other parts we find that it everywhere contains large numbers of small round cells and spindle shaped cells the small cells being those which have but recently proliferated from the fixed connective tissue cells while the spindle shaped ones are gradually taking on more themselves the characters of the cells from which they are derived.

The formation of cysts on this condition of the kidneys is to be explained by the fact that as the bands of connective tissue surround the tubules in certain portions and there partly occlude them by pressure, the portion behind the occlusion which is free becomes filled and dilated with the watery part of the urine which are still excreted, and being unable to pass the obstruction distends the tubule until it loses all resemblance to its original condition and becomes converted into a fluid containing sac. As in the early stage of this disease we saw that the muscular coats of the arteries was hypertrophied, so now in this advanced stage this hypertrophy is still more marked.

---

THE URINE:- In the early stage of the disease this may be normal in amount, but later it is increased. This increase is probably dependent upon the high arterial pressure which exists during chronic interstitial nephritis. In color the urine is pale and of low specific gravity. (from 1010 to 1015.) Albumen is usually present in small quantities and may at times be absent. The amount of urea is diminished.

DROPSY:- If present is slight and may even be absent throughout the entire course of the disease.

THE PULSE AND HEART:- The pulse is always of high tension, hard and incompressible, and in those of advanced years there is usually a marked degree of arteria sclerosis. Hypertrophy of the muscular coats of the renal vessels, as well as of the arteries generally is present. Hypertrophy of the left ventricle is also present and can usually be easily detected by percussion. These changes together with the passage of an abnormally large amount of urine of low specific gravity, light color, and containing small amount of albumen are points upon which to base a diagnosis of chronic interstitial nephritis which should be strengthened by the clinical symptoms, age and habits of the patient.

# AMYLOID DISEASE OF THE KIDNEY.

DEFINITION:- A condition of the kidney in which the amyloid substance is found deposited in various localities.

ETIOLOGY:- Syphilis, tuberculosis of the lungs or of the bones and wasting diseases generally especially if associated with collections of pus of considerable size are associated with deposits of waxy material in the kidneys as well as in such other organs as the liver and the spleen.

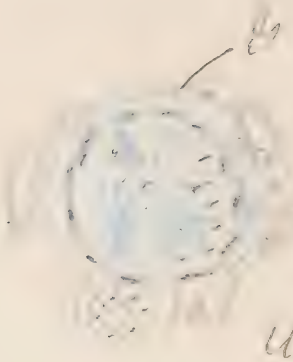
VARIETIES:- We may recognize three; (1.) in which the deposit of a waxy material seems to be the only lesion. (2.) A large waxy kidney, and (3.) A small waxy kidney.

SEATS OF THE WAXY DEPOSIT:- The amyloid substance is found to be deposited chiefly within the glomerular tufts in the walls of the vas efferens and of the straight arteries of the medulla and at times in those of the small and middle sized arteries of the intermediate zone. According to some observers the walls of some of the tubules, especially of the larger ones in the medulla sometimes show a deposit of the amyloid material.

## I. SIMPLE AMYLOID KIDNEY.

In this condition, the kidney seems to be free from all renal disease except the deposit of amyloid material in the localities just mentioned. There are very few gross appearances to mark this condition. The kidney is usually paler than normal and the cortex when treated with a solution of iodine has a speckled appearance, the small points representing waxy glomeruli.

When examined microscopically after being properly stained small deposits of amyloid are seen in the above mentioned places. The tubules appear to be in a healthy condition and there is no change to be seen in the interstitial tissue.



A = amyloid
U.T = urinferous tubule.
G = Glomerulus.

## II.- Large Amyloid Kidney.

PATHOLOGICAL ANATOMY:- At autopsy the kidney is found to be considerably enlarged and to be increased in weight. The color the organ is decidedly pale and its consistency very elastic. The capsule strips off very readily, leaving a glistening smooth pale surface. Upon section it is noted that the paleness of the kidney is general the medulla being as much affected as the cortex.

PATHOLOGICAL HISTOLOGY.- Upon microscopic examination one finds the amyloid material deposited in the portions of the organ already mentioned as the seats of the disease and in addition it is seen that there are marked changes in the tubules and interstitial tissue. These consist of a catarrhal inflammation of the tubular epithelium, a catarrhal nephritis, (see Catarrhal Nephritis) and of a diffuse increase in the interstitial tissue. (see the second stage of Catarrhal Nephritis and Chronic Interstitial Nephritis) In these cases we have probably a catarrhal and interstitial nephritis started up in an organ which was previously affected with amyloid deposits.

## III.- Small Amyloid Kidney.

The large amyloid kidney is by far the most common met with, but we also at times find a small kidney in all respects resembling the small kidney of chronic interstitial nephritis which upon examination we find to be infiltrated with waxy deposits. This form of the disease is very brief only one did cirrhotic kidney within which the amyloid material had been deposited, and for this reason it needs no further description. It is probable that if all the cirrhotic kidneys met with were stained for waxy material one would find it present to a greater or less degree in a larger proportion of them.

Some observers however look upon this small amyloid kidney as a late stage of the large kidney just described, and from the fact that in that kidney we find the earlier stages of interstitial inflammation it is not unreasonable to suppose that as the new formed tissue contracted the small amyloid kidney was produced. According to this view we would not have a contracted kidney in which the amyloid material had been deposited, but a chronic interstitial nephritis supervening in a kidney that was already affected with waxy material

---

THE URINE - The quantity is usually large and of low specific gravity. It also contains albumen and casts are rare, when present they are of the hyaline variety

DROPSY:- In the simple form is slight or absent. In the large amyloid kidney it is at times quite marked.

---

# GLOMERULO-NEPHRITIS

DEFINITION:- Inflammatory conditions affecting the various glomerular structures

VARIETIES:- We may recognise three chief varieties of inflammatory condition of the glomerular structures: (1) An acute catarrh of the capsular space. Acute Catarrhal Glomerulo-Nephritis. (2) An acute interstitial inflammation of the glomerulus Acute Interstitial Glomerulo-Nephritis, and (3) a Chronic Interstitial Glomerulo Nephritis

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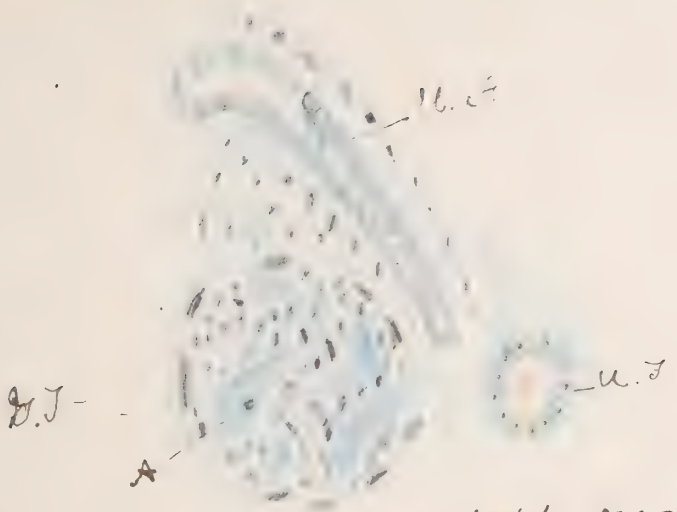
## ACUTE CATARRHAL GLOMERULO-NEPHRITIS

PATHOLOGICAL HISTOLOGY - The chief lesion of this condition is confined to the epithelial cells which line the inner side




U. T = uriniferous tubule.
G. T - Glomerulus (tuft).
A - Amyloid material.
U. C [illegible]
[illegible]

of the glomerular capsule so that in time there is left to each one a tuft that is functionally greatly impaired. These undergo great proliferation to such a degree that the glomerular tuft is seriously injured and its function consequently greatly impaired. The proliferated cells become flattened out by their mutual pressure so that in one section one at first takes them for fibrous tissue cells - a closer study of several glomeruli however will show that they are in reality flattened cells derived from the inner lining of the glomerular capsule. In perfectly typical cases of this disease this is the only lesion of importance, but in most cases there is an accompanying catarrhal inflammation of the tubules. Acute glomerulo nephritis is very frequently associated with scarlet fever.

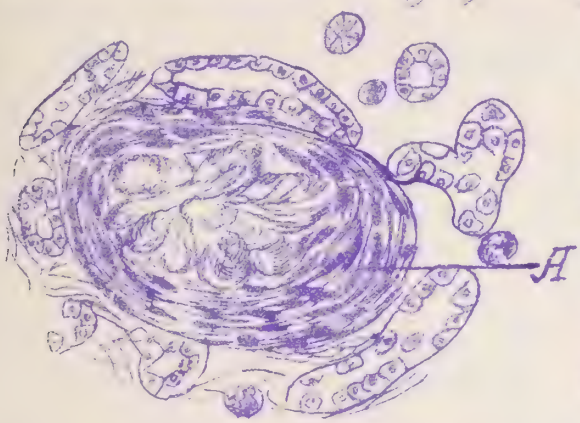



FIGURE 22.

Acute Catarrhal Glomerulo-Nephritis

Showing (A) Proliferation of Cells lining Bowman's Capsule

## ACUTE INTERSTITIAL GLOMERULO-NEPHRITIS

DEFINITION - An acute interstitial inflammation confined to the glomerulus.

PATHOLOGICHL ANATOMY - The kidney at autopsy is considerably larger than normal and to the quite extended. The capsule strips very easily. Upon section the organ is seen to be most marked in the medullary portion. The cortex is thickened and this thickening accounts for the increase in the size of the organ. On section the cortex is greyish red here and there, where it is examined closely with the naked eye are seen little rounded spots which represent the glomerule.

PATHOLOGICAL HISTOLOGY:- When the glomeruli are studied with the microscope it is seen that the space which normally exists between the tuft of glomerular vessels and the capsule is entirely obliterated. The whole space within the capsule is diffusely infiltrated with small round cells These lie not only in the capsular space, but also between the capillaries of the tuft. The general impression given is that the whole glomerulus has been stuffed with small round cells. In the majority of cases the glomerulus alone is the seat of this small celled infiltration, but in a small number of cases a few similar cells may be seen immediately about the glomeruli. Rarely if ever is the general interstitial tissue of the organ affected.

The origin of these small round cells has been variously stated. Klebs considered that they were derived from the slight tissue that lies between the glomerular capillaries. Others have considered that they were derived from the cells of the capillary vessels themselves while still another view is that they are simply leucocytes which have escaped from the capillary vessels of the glomerulus. It is probable that they are derived from all of these sources and not from any one alone

The tubular epithelium is usually found to be somewhat swollen and desquamated, and in some instances to be fatty the changes however are not nearly so marked as in true catarrhal nephritis and are probably the result of the general disturbance in the kidney nutrition.

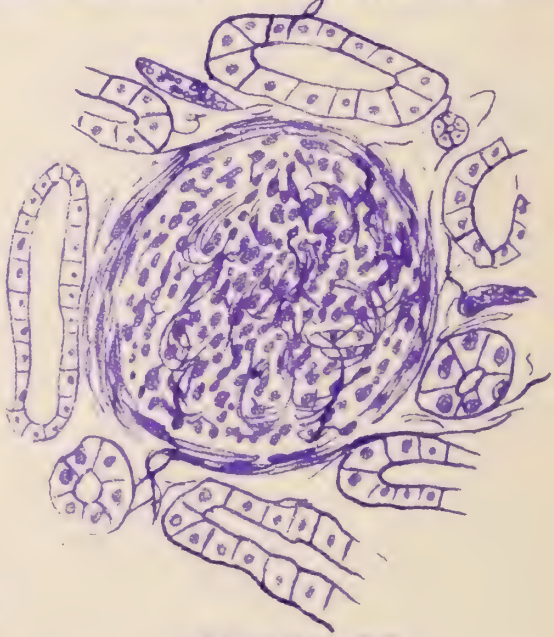


FIGURE 23.

Acute Interstitial Glomerulo-Nephritis.

Showing proliferation of cells within the glomerular tuft.

Like the simple variety first described this form of

glomerular inflammation, is frequently the sequel of scarlet fever. From the location of the morbid changes it is of necessity a dangerous condition. Both forms just described are more often found in childhood than in middle or advanced life.

THE URINE - Is usually smaller in amount than normal and is of low specific gravity. It contains much albumen and is frequently bloody.

## CHRONIC INTERSTITIAL GLOMERULO-NEPHRITIS.

When death from the acute variety does not result within a short time the disease runs a more chronic course, and the usual changes found in chronic inflammation of fibrous tissue are found to have occurred in the mass of young proliferated cells which we saw in the acute interstitial variety. All the young cells which are at first round, gradually develop into connective tissue fibers, destroying thereby all remnants of the glomerular structure until finally the place of the glomerular tuft is occupied by a mass of connective tissue. In rare cases there seems to be complete recovery from the acute variety.

# TUBERCULOSIS OF THE KIDNEY

VARIETIES - Two are met with: (1) An acute miliary form and (2) A chronic caseous variety.

## THE ACUTE MILLIARY FORM.

PATHOLOGICAL ANATOMY - This form of the disease is always associated with an eruption of miliary tubercles on other organs, it is part of a general miliary tuberculosis and may be found in some cases of advanced pulmonary tuberculosis, where there are similar lesions in the liver

and spleen, though the kidney seems to be less frequently affected in this way than either of these two organs.

In general the tubercles are found as small millet seed nodules scattered in largest numbers in the cortex and mostly just below the capsule. Frequently when the capsule is removed these are visible to the naked eye as small semi-translucent specks on the surface of the organ. When the tubercles are of any size they can be seen on section as yellowish masses very sharply defined from the surrounding kidney tissue. When found in the medulla they are usually elongated in shape.

PATHOLOGICAL HISTOLOGY:- The histology of tubercle has already been given, and as these bodies in the kidney differ in no respect from tubercles in other parts a description of them will not again be given. In some books it is stated that giant cells are comparatively rare in this form of kidney tuberculosis. The author's experience however has been quite the contrary and in specimens given for study it will be found that these cells are decidedly more numerous than in most specimens of tuberculosis from other organs. The tubercles originate within the minute vessels. This is what one would naturally expect seeing that the tubercle bacilli are brought to the organ by the blood and become lodged in these small blood passages. What may be mentioned as rather distinctive of this form of renal tuberculosis is that these tubercles rarely become sufficiently softened to break down into particles, except of the very smallest size; this probably due to the fact that the patient dies before time enough for this destructive process to set in has elapsed

A

# LABORATORY MANUAL

— of —

# Pathological Anatomy.

## FOURTH PART.

## DISEASES OF THE INTESTINES

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Tuberculosis of the Intestine . . . . . " 114

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# THE INTESTINES.

For the normal anatomy and histology of the stomach and intestines consult works on anatomy and histology.

## TYPHOID LESION OF THE INTESTINE.

The chief lesions in typhoid fever as found on the intestines are located in the ileum; occasionally they will be found in the upper end of the colon about the ileo-cecal valve, but this is rare; still rarer is it to find them on the jejunum and stomach though cases have been reported in which in these localities there was evidences of the disease.

STRUCTURES AFFECTED:- Scattered throughout the intestines there are numerous collections of adenoid tissue little gland-like bodies which lie in the mucosa and extend somewhat deeply into the sub-mucous coat. Within the ileum these gland-like bodies are particularly numerous and occur in two distinct forms. One of these, the so-called solitary follicles are simple and small collections of lymphoid cells scattered promiscuously throughout the mucous membrane, the other the so-called Peyer's patches are nothing more nor less than accumulations of the solitary follicles, forming masses of adenoid tissue of considerable size, usually oval or oblong in shape with their long axis in the long axis of the intestine. These solitary follicles and Peyer's patches are the seat of the lesions in typhoid fever.

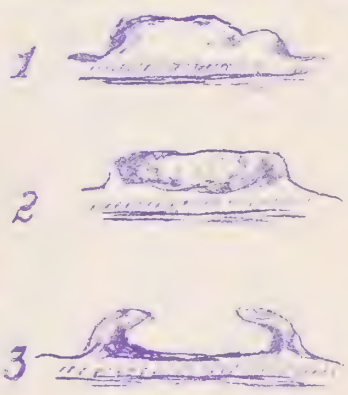



FIGURE 24.
Diagram to represent stages of Typhoid Lesions in Peyer's Patches.
(Modified after Thoinot.)
1. Early stage - tubercle formation.
2. Later. Sequestrating sloughing.
3. Later formation. Ulcer with crater shaped edges.

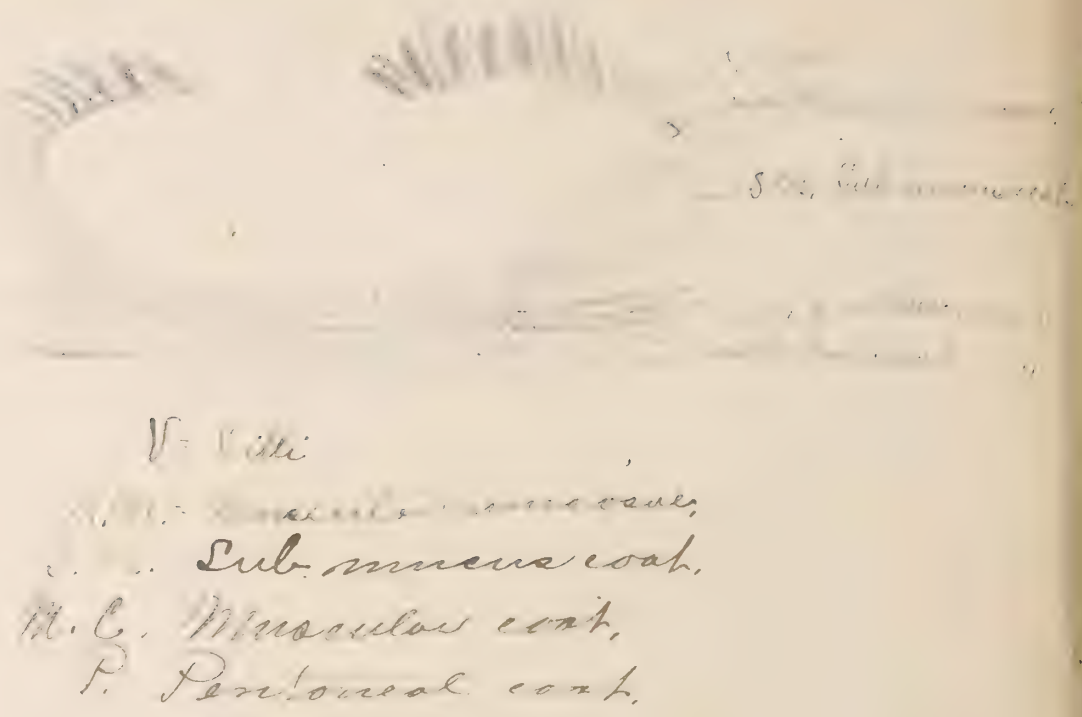
PATHOLOGICAL ANATOMY:- In the earliest stages of the disease the glandular structures above mentioned are simply swollen;

they project visibly above the surrounding surface and may be surrounded by a zone of congestion. The swelling is due to the fact that these bodies have become the seat of a small celled infiltration but though they are swollen and prominent they do not feel hard but to the fingers quite soft. The cellular infiltration in these cases is probably of an inflammatory nature and is due to the presence of the typhoid bacillus, which even in this early stage of the disease can be demonstrated in the swollen glandular structures. After ten days to two weeks the infiltrated glands begin to slough and leave the ulcers which are characteristic of this disease. This sloughing is due to the fact that the cells which have gathered in such large numbers within the solitary follicles and Peyer's patches become necrotic and broken up into a mass of ill-defined granular material. Their nuclei cease to stain

THE ULCER:- The ulcer which is produced by this process of necrosis and sloughing has the following characteristics.

1. If it be a solitary follicle which has sloughed the ulcer will be more or less round in shape, but if it be a Peyers patch the ulcer will be oval, with its long axis in the long axis of the intestine
2. The floor of the ulcer is generally the muscular coat of the intestine and is comparatively smooth, though occasionally there will be a few shreds of the mucosa or sub-mucous coat left clinging to it.
3. The edges of the ulcer are usually unattached, that is the ulcer has undermined the mucous coat, and the edges can be floated up on water
4. Where the ulcer has not eaten into the muscular coat the serous coat of the intestine will be found to be unaltered no such thickening being noticed as is seen in cases of tuberculosis of the intestine.

When perforation has taken place it is found that the ulcer has eaten through all the coats of the intestine, the sloughing process not ceasing when the muscular coat


V. Villi
Sub. mucous coat.
M. C. Muscular coat.
P. Peritoneal coat.

have been reached as is usually the case. In these cases death follows rapidly from peritonitis.

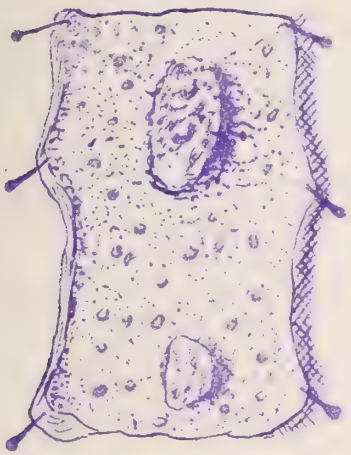


FIGURE 25

Typhoid Fever. Ileum.

Showing infiltration of Peyer's patches and solitary follicles.

Where the case of typhoid fever progresses to recovery and the patient dies some time later from some other disease we find upon examining the intestines that there is no evidence whatever of the previous attack of fever. The ulcers therefore heal without the formation of a cicatrix and there is in consequence no constriction of the intestine from cicatricial contraction as occurs in tuberculosis

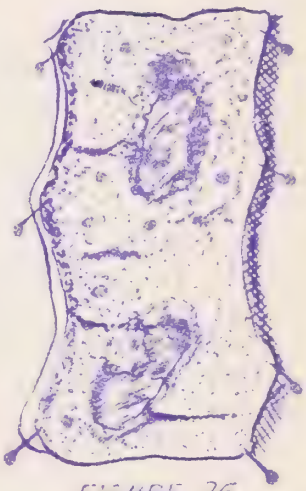


FIGURE 26

Typhoid Fever

Showing Ulcer and Peyer's patches

ETIOLOGY. The cause of this disease is a micro-organism discovered by Klebs, Eberth and Koch about the same time (1880-1881). It is a short bacillus which does not form spores and which is actively motile. When sought for within the body it can be found in the intestinal sloughs, and in the intestinal discharges, though in the latter with some difficulty. Within the glandular structures which are infiltrated it can be found in large numbers. It can be found also within the mesenteric glands, in the liver, spleen and kidneys. It is not easily found in the blood.

# TUBERCULOSIS OF THE INTESTINE

Tubercular disease of the intestine occurs either as tuberculosis of the mucous membrane, or of the serous coat lining the outside of the gut. The former is almost always the result of advanced tuberculosis of the lungs in the stage of cavity formation, while the latter is a part of a general Peritoneal tuberculosis. Very rarely there is seen a primary tubercular infiltration of the mucous membrane. This generally occurs in children and is the result of eating food that is infected with large numbers of tubercle bacilli.

TUBERCULOSIS OF THE MUCOUS MEMBRANE:- The chief seat of this form of the disease is the cæcum, from this point it diminishes upward and downward, sometimes in severe cases it may be met with as far down as the rectum. Generally the first evidences are met with on the ileo-cæcal valve where it takes the form of numerous hard, raised yellowish projections which are infiltrated lymph follicles. These become caseous at their summit, and when ulceration begins the caseous summits soften and small volcano-like pits result.

These may be widely separated but when they are close together they frequently fuse and form ulcers of considerable size.

THE ULCERS:- When we examine these excavations closely we find that they have the following characteristics:

1. Their floor are formed of the sub-mucous coat which is greatly infiltrated with tubercular nodules and is quite rough and necrotic.
2. The edges of the ulcer are not regular as are those of typhoid fever, but are sinuous, infiltrated, and quite hard to the feel. They are not as a rule undermined as is the typhoid ulcer. The hard edge is caused by

tubercular infiltration, and when we examine it with the microscope we find numerous small tubercles here and there in the border of the ulcer in various stages of degeneration. It is by the breaking down of these tubercles that the ulcer increases in size.

3. As a rule the long axis of the ulcer is transverse to that of the intestine, though the ulcer may be round.
4. The tubercular ulcer rarely perforates.
5. Opposite the base of the ulcer the serous coat will be found to be considerably thickened and infiltrated. To the naked eye this has the appearance of a stellate milky patch. From the centre of this patch one can see tubercles radiating in all directions following the course of the lymphatics in the serous coat.

FIGURE 27.
Tubercular Ulcer of intestine

6. Below and about the ulcer there is considerable small round celled infiltration, frequently running through the muscle coats into the serous coat. Here and there both in the submucous and in the muscular coats can be seen small tubercles frequently with giant cells.

This form of the disease then begins in the retiform lymphoid tissue which absorbs the tubercular poison and within which there are developed small tubercles which following the general rule for tubercles develope to a certain point and then begin to undergo degenerative changes resulting in caseation, softening and the formation of ulcers. These ulcers must

heal, for within their edges there are constantly new tubercles being formed which by breaking down enlarge the ulcer. Here and there at times a partial healing may take place and it is not infrequent for a stricture of the bowel to result from such cicatricial contraction.

FIGURE 28

Portion of tubercalous ulcer showing infiltration of edge of ulcer, thickening of serous coat below ulcer and tubercle in serous muscular and sub-mucous coats.

## TUBERCULAR AND TYPHOID ULCERS COMPARED.

| TYPHOID ULCER. | TUBERCULAR ULCER |
|---|---|
| 1. Edges Undermined. Can be floated out of water; thin and composed of mucosa and sub-mucosa. Usually red in color. | Edges not undermined; thick, nodulated, sloping, Infiltrated with small tubercles. Pale, or red in color. |
| 2. Long axis usually in the long axis of the intestine. | Long axis frequently transverse. Not characteristic. |
| 3. Floor smooth and vascular. | Floor roughened with tubercular nodules. Avascular, with yellow cheesy spots. |

| | |
|---|---|
| 4. No thickening of the serous coat | Deposit over base of ulcer thickened, yellowish, with small tubercles radiating in all directions along lines of the lymphatics |
| 5. Mesentery unaltered. Glands enlarged, soft and pink | Mesentery thickened at its attachment to bowel. Glands enlarged firm and sometimes caseous |
| 6. Perforation not uncommon, producing peritonitis, Hemorrhage not uncommon. | Perforation, peritonitis and hemorrhage all rare |
| 7. Extension takes place both laterally and in depth. | Extension takes place usually laterally. |
| 8. Heals by granulation. | Very rarely heals |
| 9. Does not cause constriction when it heals. Seldom breaks out afresh | Frequently causes constriction of the bowel when it heals. Often breaks out afresh. |
| 10. Microscopically: a specific inflammation of the adenoid tissue; blood vessels distended. Dense masses of small round cells in mucosa and submucosa. Line of demarcation with resulting abscess beginning in the solitary glands and in Peyer's patches. | Microscopically: a specific inflammation of the adenoid tissue and of the mucous membrane ending in caseation and connective tissue formation. Formation of typical tubercles in the mucosa and submucosa and frequently in the muscular and serous coats. Small round cell infiltration very well marked. |

# TROPICAL DYSENTERY

## (AMOEBA COLI DYSENTERY)

DEFINITION:- A form of dysentery found in tropical countries and occasionally in the United States, which is frequently associated with abscesses of the liver.

ETIOLOGY - An animal parasite or amoeba known as the amoeba coli. This organism is found in the coats of the intestine, in the intestinal discharges and in the contents of the liver abscess.

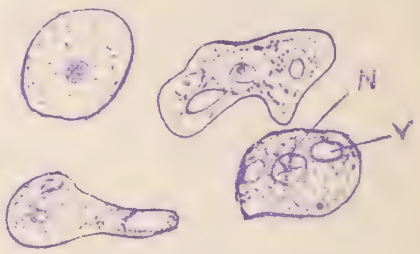



FIGURE 29

Amoeba Coli from Stools of tropical dysentery.
N. Nucleus. V. Vacuole.

GROSS AND MICROSCOPICAL CHANGES - The disease usually begins and is confined to the large intestines, though according to some observers the ileum is occasionally involved. In the early stages there is much congestion of the vessels of the serous coat as well as of the mucosa which is frequently of a deep purplish color. Probably the most striking gross appearance is the great thickening of all of the coats of the intestine and especially of the submucosa which is increased in size not only on account of its edematous condition, but also by sharply circumscribed nodular accumulations of cells in which there are frequently small cavities filled with degenerated tissue, communicating with the surface of the mucosa by small openings. In more acute cases these cavities in the mucosa extend deeper down as sinuous tracts by the discharge of their contents [illegible] and either small ulcers

FIGURE 30

Amoebic Colitis. Dysentery. Ulcer with undermined edges. a Mucous membrane. b Submucous tissue. c Muscular coat. d Muscular coats. E Serous coat.

frequently opening at several points, with extensive sloughs advancing to the sides. A third form of ulcer sometimes seen which has very slightly undermined edges, representing simply excavations in the thickened submucous tissue. The mucous membrane seems to suffer least, and the lesions on it are chiefly secondary to those in the submucosa. "The process is essentially one of advancing infiltration and softening in the submucous and intermuscular tissue with following necrosis of the overlying tissue." A little distance away from the ulcers there is little or no change in the mucous membrane, and even in cases where it is extensively undermined it may show no changes.

In the bases and about the margins of the ulcers there are large numbers of small round cells, and lying in the tissues beneath the floor of the ulcer one may find in all cases a varying number of the amoeba with which this disease is associated. In addition there will also be seen a number of rather large cells looking very much like the amoeba, though somewhat smaller which are probably dilated and enlarged connective tissue cells. The amoeba themselves are large round, or elongated cells, with vesicular nuclei in granular protoplasm which frequently contains one or more vacuoles.

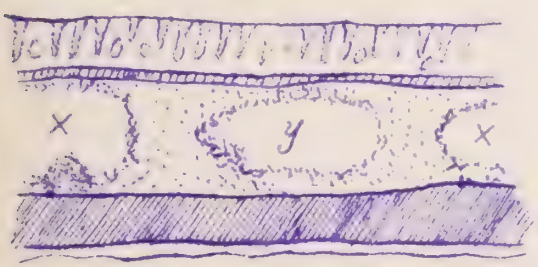



FIGURE 31

Tropical Dysentery. Showing a part of two ulcers (x, x) and an undermining passage in submucosa (y).

In the walls of the ulcers the vessels are very much distended and about these the accumulation of small round cells is very marked. If recovery take place in these cases the mucous membrane is never restored and years after may be found in a shrivelled and tattered condition.

Liver abscesses of considerable size are frequently associated with this form of dysentery and it is not an infrequent occurrence for these, when situated in the upper portion of the liver, to break through the diaphragm, and thus [illegible]

through the lung. In these cases the amoeba coli are to be found in the sputum and upon their presence a diagnosis can be made.

# CROUPOUS ENTERITIS

SYNONYMS:- English Dysentery. Cholera Nostrans.

DEFINITION:- A croupous inflammation of the mucous membrane of the large intestine.

GROSS PATHOLOGY - The mucous membrane of the large intestine is found to be injected and of a dark purplish red color. Here and there can be found a number of small superficial ulcers in the mucosa. Over the surface in many places there is what appears to be a false membrane which is of a dirty gray color and very easily removed by scraping.

MICROSCOPICALLY - When one studies sections of the intestine in this condition one finds that the gray membranous like covering of the intestine, which at first would be taken for a false membrane composed of fibrin, contains very little if any fibrin and is for the most part really a superficial sloughing of the mucous membrane. In some cases however though in few, there is a distinct false membrane of fibrin over the surface of the diseased mucosa, but in most cases the necrotic material seen is the remains of the mucous membrane with its contained crypts which has lost its vitality, become fatty and been cast off.

Most of the vessels of the intestine, but not those of the peritoneal covering are engorged with blood, and here and there in the submucosa and between the bundles of fibers in the

muscular coats can be seen extravasations of blood and at other places collections of small round cells.

TERMINATIONS:- Where the disease does not cause death as it does in the majority of cases, there is frequently found a healing of the wounded parts accompanied by extensive cicatricial contraction which may in some cases lead to a great narrowing of the intestinal canal. The scars may be so numerous that but little of the mucous membrane is to be seen.

# TUBERCULOSIS OF THE PERITONEUM.

## (TUBERCULAR PERITONITIS.)

DEFINITION:- An inflammation of the peritoneum caused by the presence of tubercle bacillus and characterised by a wide spread eruption of milliary tubercles.

ETIOLOGY:- The immediate exciting cause is of course the tubercle bacillus, but how this gains access to the peritoneum is in many cases unknown. In many cases it is supposed to be derived from the intestine.

GROSS PATHOLOGY:- At autopsy the whole peritoneum is found to be injected and in a state of sub-acute inflammation. Scattered over its entire surface there are innumerable small yellowish or translucent nodules varying in size from a pins head to that of a pea. The larger ones are usually yellowish in color and on section are distinctly caseous. These masses are the tubercles. Great fibrous adhesion of the intestine to adjacent parts and to the parietal peritoneum is usually found and in some cases it is only with difficulty that the intestines can be removed except as one mass. The tubercles are also found scattered

over the capsule of the liver, but are not found in its substance. They are also found scattered over the under side of the diaphragm where they have evidently developed within the large lymphatics. Other organs throughout the body may or may not be tubercular.

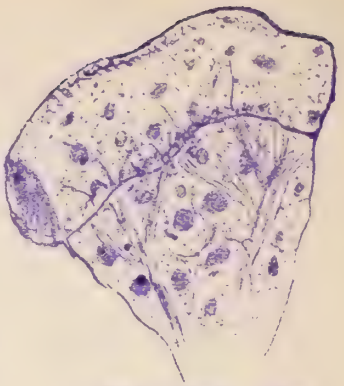


FIGURE 32.

Portion of intestine with Mesentery attached showing tubercular nodules

This disease is entirely different from the tuberculosis of the intestine already described where the mucous and sub-mucous coats are the chief seats of the disease. In these latter cases the peritoneal coat is usually free from tubercles except just over the bases of the ulcer in the mucosa. Likewise in the great majority of cases of primary tuberculosis of the peritoneum there is no tubercular disease of the mucous membrane.

MICROSCOPICALLY.- These tubercles differ in no respect from miliary tubercles in other situations and as their structure has already been given in other places it will not be described here.

The End

1

2

3

# BRIEF NOTES

ON

# The Conduct of Post-Mortem Examinations.

BEING

## APPENDIX, A.

TO

## A LABORATORY MANUAL

OF

## PATHOLOGICAL ANATOMY

BY

DELANO AMES; A. B. M. D.

BALTIMORE, MD.

1897.

BALTIMORE:
Printed by C. A. HASKELL,
1 S. Calvert Street.

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# The Technique of Post-mortem Examinations.

## Chapter I. Introduction

I. The report of a properly conducted post-mortem examination makes, in many cases, a valuable and important document. On the other hand an incomplete report of a poorly conducted autopsy is worse than useless as it not only gives no valuable information, but may even lead to the formation of erroneous conclusions. An investigation into the condition of a body after death is usually made for one of three purposes, either in the hope that the result of the examination will throw light upon obscure conditions, produced by the disease from which the individual died, and thereby widen the scope of our knowledge of the morbid changes which occur within the economy; or for the purpose of demonstrating pathological changes to students of medicine; or else the investigation is made at the instance of the State, in order that certain facts relative to the cause of death of the individual may be ascertained, upon which, not infrequently, rests the guilt or innocence of an accused party.

It can be readily seen therefore, that the opening statement just made, is not only strictly true, but that upon every one called upon to conduct a post-mortem examination there rests a degree of responsibility, that in certain cases may well render the operator extremely careful in his work.

In this country, where it is the exception for the State to appoint regularly qualified autopsy physicians, and to lay down certain rules and regulations for their guidance in conducting medico-legal autopsies, and where in consequence it frequently falls to the lot of the general practitioner to make such examinations, it behooves every one to fit himself as completely as possible for this oft-time exceedingly responsible duty.

The short account given in the few pages that follow, of the usual methods adopted in conducting an autopsy, will it is hoped be sufficient to guide the student in his work, it being understood that within the compass of short notes of this kind only the chief essential details can be pointed out, and the general lines of procedure laid down. Those who wish to go deeper into the subject and to thoroughly fit themselves for this sort of work are advised to consult larger works on the subject, a short list of which can be found at the end of this manual.

It is needless to say that every one before attempting to do autopsy work should be well grounded in normal anatomy, and be possessed of an accurate knowledge of the appearance of healthy organs and parts. In addition to this essential requirement, accuracy of observation, even to the smallest detail should be strenuously cultivated, for nowhere is the power to observe accurately more called for than while endeavoring to discover the various changes that disease or injury have produced in a body brought to one for examination.

To the above qualifications requisite to satisfactory post-mortem work may be added the ability to accurately and forcibly describe the conditions that are observed, so that any one reading the report of the autopsy would have presented to his mind accurate pictures of the various changes met with. Too much, otherwise good work is seriously marred by being carelessly recorded.

The best method for recording the results of a post-mortem examination will be given on a subsequent page. It should be remembered however that especially for the beginner it is important that every thing observed, whether it appear to him to be of importance or not should be carefully recorded, as it is only by increased experience that one can judge of what is and what is not important. A further end is also gained by systematically recording all that is observed, one forms the invaluable habit of observing, and observing closely

and systematically so that in the end very little is apt to be overlooked. This habit, once established, is never lost. It should also be remembered that all normal conditions should be recorded as such, and the future reader of the report not be left to infer that parts were normal because they were not put down as being altered in any way.

Were the autopsies so frequently made in all our hospitals today carefully done and systematically recorded a large amount of valuable pathological material would be saved, and it is a fact very much to be regretted that "the wanton waste of valuable pathological material in many hospitals and public institutions at the present time on account of defective examination and imperfect record keeping reflects seriously upon the scientific interests and spirit of the attending medical and surgical staffs"*

At the time of making an autopsy the operator should dictate notes of such completeness that at some future time a full and readable account of the various conditions found could be made. For the purpose of making the preliminary notes in a regular order at every post-mortem, many institutions supply printed blanks that are filled out at the time of the autopsy and from which the future report is compiled. In many instances these blanks when filled out form a sufficiently full report of the case and can be filed away in the hospital attached to the clinical records of the case. At other times, especially where a medico-legal report is to be made, the report made at the time of performing the autopsy should be as complete as is necessary, and this original document, signed by the one who performed the autopsy and dictated the notes and by the one who wrote the record should be presented to the Court, no subsequent additions, or alterations being made.

---

* "Post-Mortem Technique". McFarland.

II. Before describing the various steps to be followed in conducting the post-mortem examination it will be best to describe the instruments and other appliances that should always be on hand.

## Instruments.

Knives:— 1. A section knife as first described by Virchow. This instrument should be run at about 24 centimeters in length, of which 9 to 10 centimeters should belong to the handle, and the blade being stout, broad and well rounded toward the point. (Fig. 1) The handle should be such as can be readily grasped in the palm of the hand, for it should be understood from the start that in making post-mortem examination the knife should never be held as one holds a pen, between the thumb and fingers, but in such a way that when the arm is extended the knife appears as a direct prolongation. By holding the instrument in this way clean, long incisions can be made by drawing the knife firmly over the parts with a full arm movement.

2. A strong ordinary cartilage knife. This is chiefly used to sever the costal cartilages and to do other heavy work.

3. One or two ordinary dissecting scalpels to be used for the finer dissections.

4. A long, thin, broad bladed knife to be used in making sections of the brain and other large organs. (Fig. 2.)

Scissors:— 1. A pair of small probe pointed scissors to be used in opening vessels and small canals.

2. A pair of medium sized ordinary scissors.

3. One enterotome, (Fig. 3.) for opening the intestines, etc.

Measures:— One brass measure 20 centimeters long, divided into centimeters and millimeters.

One measuring glass holding 300 cubic centimeters for measuring the amount of fluids found in the various cavities of the body.

A set of graduated cones, (Fig. 4.) for measuring orifices, etc.

Two or three pairs of dissecting forceps.

FIG. 1. Section Knife. (Virchow's.)

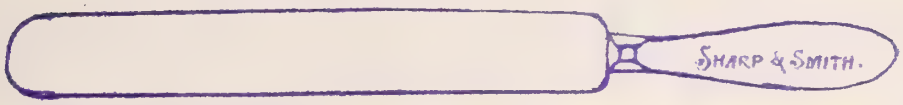


FIG. 2. Long Flat Double-Edged Knife for Sectioning the brain.

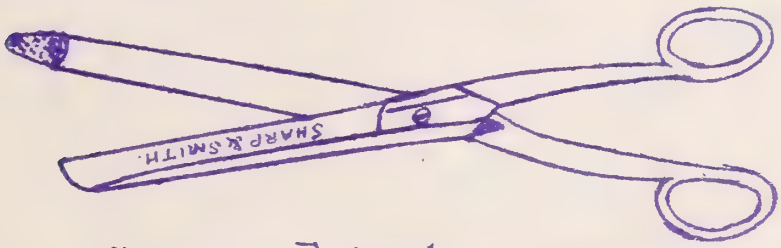


FIG. 3. Enterotome - (Proper Shape)

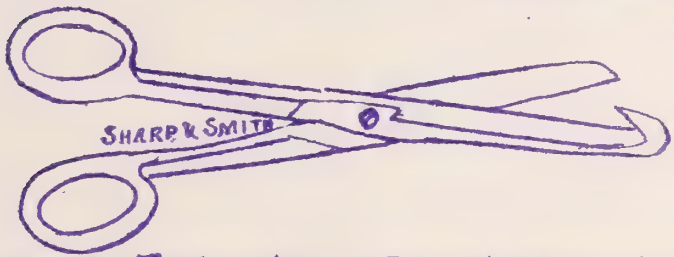


FIG 3' Enterotome – Improper Shape because the hooked end of the longer blade constantly catches in the tissues.

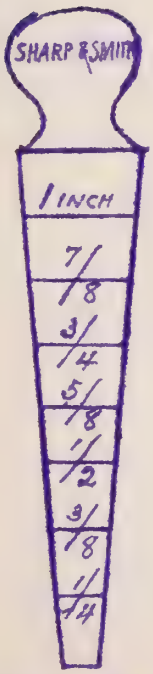


FIG. 4. Graduated Cone used to measure the Size of orifices.

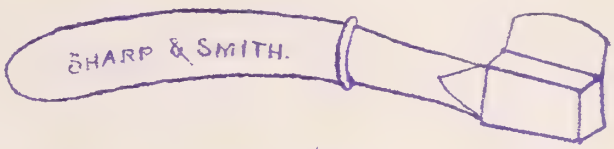


FIG. 5. Bone Chisel. (Rachitome).

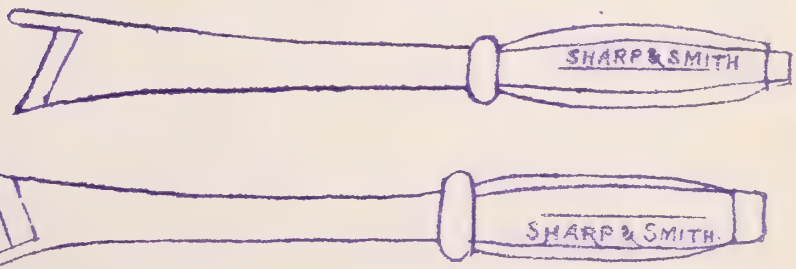


FIG. 5' Brunetti's Bone Chisel, (Left & Right.)

A pair of scales and metric system weights, from 5 kilograms down.

A stout mallet.

One or more bone chisels

One bone saw. Or an ordinary butchers saw will do.

One pair of bone forceps

Several post mortem needles and stout linen thread.

Glass or porcelain dishes, jars, sponges, plates, magnifying glass, etc.

## Reagents

III. It is always necessary, when one expects to save tissue from an autopsy, to have on hand in quantity, one or more preserving agents, into which small portions of tissue can be placed to harden for future examination. Of such reagents the ones mostly used and which give the greatest satisfaction are:-

Alcohol:- This should be used where the tissues are to be examined for micro-organisms and should be either absolute in strength or 95%. Alcohol however dissolves fatty substances and the coloring matter of the red corpuscles, and where it is desired to save these it is best to use the following:-

Formol:- This is a solution of formic aldehyde in water and is found in the market as a 40% solution* which before use must be reduced by the addition of water to 5% strength. This reagent has the advantage of being cheaper than alcohol, and it also hardens tissue more rapidly and with less shrinking. I now use formol entirely, and it is my custom to allow small pieces of tissue to remain in this solution for a few days, after which they are removed, washed, and transferred to 80% alcohol where they remain till needed for examination.

For preserving portions of the brain and nervous system

* It also appears under the trade name of "Formalin"; and also as "Form-aldehyde".

It is some[times] advisable to use what is known as Müllers Fluid, although Formol can also be used, which is cheap and easily made.* A disadvantage of this reagent is that at first it must be renewed every day. When care is taken to change the fluid very often entire organs may be perfectly preserved.

Except where large portions of the tissue or whole organs have to be saved for purposes of demonstrating gross pathological conditions, only very small pieces should be taken as these harden more rapidly and with less shrinking, and from a very small piece of tissue a very large number of sections can be cut.

IV. If it is desired to make cultures from the fluids and organs of the body, one must have on hand a supply of culture tubes and platinum probes. For the technique of making cultures, and their subsequent care and study, the reader is referred to some work on bacteriology.

## Chapter II.

### The General External Examination.

V. Before beginning any post-mortem examination, whether upon the body of one who has died under observation in a hospital, or in private practice, and about which practically everything that is of importance is known, or upon a body presented for autopsy for medico-legal purposes, the first thing to be done is to make a careful inspection of the cadaver and to record the same in as concise a manner as possible.

For purposes of convenience and to insure the completeness of the examination it is well to follow some definite order, and the following, or some modification of it may be employed.

---

* Müllers Fluid is composed of the following ingredients

| | |
|---|---|
| Potassium bichromate | 2½ parts |
| Sodium sulphate | 1 " |
| Distilled Water – By weight | 100 " |

1. Sex.
2. Race.
3. Apparent age.
4. Height.
5. Condition of nutrition.
6. Presence of Rigor Mortis.
7. How long dead
8. Body Temperature
9. Post-mortem lividity

## THE HEAD.

1. Color of hair.
2. Ears, discharge from, or foreign body in.
3. Nostrils, discharge from, character of
4. Mouth " " " "
5. Color of mucous membranes
6. Condition of teeth and gums
7. Wounds, if present, character of
8. Any peculiarities present

## THE NECK.

1. Goitre, wounds, or enlarged glands

## ARMS AND HANDS.

1. Oedema
2. Condition of the joints
3. Deformities, tattoo marks, etc.

## THE CHEST.

1. Development of.
2. Nutrition of.
3. Wounds, or scars of.
4. Condition of the breasts (female)

## THE ABDOMEN.

1. Shape of
2. Markings of, (linea albicantia)
3. Wounds, or scars of

## FEET AND LEGS.

1. Oedema.
2. Condition of joints
3. Deformities, scars, character of, etc.

## THE GENITALIA.

1. Development of.
2. Abnormalities of
3. Discharge from (microscopic exam of)

VI. Respecting certain of the observations above mentioned a few words of explanation will not be amiss.

For example when wounds are found they should be examined for the direction in which they were probably inflicted. In suicidal wounds for example the direction is usually from left to right. An opinion as to whether a wound was probably self inflicted or not, should be made. Again bullets make different wounds of entrance and exit, which can be determined by the inversion, or eversion of the skin

margin, and frequently by the presence of particles of clothing in the wound of entrance if the wound be in a covered part.

It is not infrequently necessary to be able to state whether a wound has been inflicted before or after death, and the following table gives the chief characteristics of wounds that have been received some time before and some time after death.

| Wounds made during life | Wounds made after death. |
|---|---|
| Edges everted due to the elasticity of the skin. | Edges of the wound in contact |
| Free haemorrhage with effusion of blood into the surrounding tissues | Little or no haemorrhage and no infiltration of surrounding tissues. |
| Clots in the vessels cut, or in the wound | No coagulum in the wound. |
| Signs of inflammation, healing or of gangrene | No such signs. |

VII. Observations such as those upon the color of the hair, the presence of tattoo marks, peculiarities of structure, etc are chiefly of value for purposes of identification.

VIII. Rigor mortis which is due to post-mortem coagulation of the myosin of the muscle fibers usually commences within five or six hours after death and passes off in about twenty four hours, though it may persist longer. The disappearance of rigor mortis is due to beginning decomposition, so that whatever will favor this change will hasten the disappearance of post-mortem rigidity. Where death has been preceded by convulsions rigor mortis comes on quickly, but lasts only for a short time. This is especially well shown in cases of strychnine poisoning and tetanus.

IX Post-mortem lividity, so frequently noticed in dependent

portions of the body, is due to the transudation of blood serum and coloring matter from decomposed red blood corpuscles, from the vessels into the surrounding tissues. It is never noted in parts that come into actual contact with the bed. This lividity must be carefully distinguished from the discoloration produced by blood extravasation due to rupture of a vessel. An incision into the skin will in the latter case show the presence of blood clot.

---

NOTE:- It is sometimes important to be absolutely certain that a body is lifeless. The following signs may be relied upon as showing that life is extinct.

Absolute cessation of the hearts action and of respiration. Where for any reason there is doubt as to whether the heart is still beating it is only necessary to apply a tight ligature to one of the fingers, when if blood is still circulating, congestion beyond the point of ligation will be produced.

Dulling of the cornea is a sign of death.

Rigor mortis which disappears when the limbs are forcibly flexed or extended is also a sign of death.

---

# Chapter III.

## The General Internal Examination.

### Order of Procedure.

X. In the present chapter we will begin the consideration of the general order of procedure, the method by which the body is opened, the various organs removed, and after removal sectioned for examination. The method really constitutes the technique of the autopsy and is of importance because organs faultily removed, will, as will be seen later, be useless either because they have been altered in the act of removal, or by being removed at the wrong time, or in the wrong way will have rendered the appearance of other organs

afterward removed so different from what they were that one is likely to be led to erroneous conclusions.

"In a systematic and scientific performance of an autopsy nothing is more difficult, and at the same time more important, than the insight into the reasons for pursuing a definite order of sequence in every detail of the examination.*** The course of the examination is usually dependent upon the order of succession. If for ordinary cases we require a definite process and a prescribed order, we do so not merely because such an arrangement is the surest guarantee for the completeness of the examination, and the best preventative against omitting important parts, but for the still greater reason that an unmethodical plan is the greatest obstacle to the subsequent collection of valuable reports. An unmethodical examination artificially and prematurely obliterates the existent condition of the parts." *

XI. The First Incision.

Standing upon the right side of the body, which should rest upon a metal, or stone topped table of such a height that the physician is not required to stoop over too far, and which is supplied with central drainage, the operator taking the section knife firmly in his hand, makes at a single, steady stroke a cut extending from the centre of the notch in the upper border of the Manubrium Sterni to the Symphisis pubis, passing to the one side or the other of the umbilicus. This cut, in the region of the chest, passes as deeply as to the bone itself, while over the abdomen it only severs the skin and subcutaneous tissue, not passing through the fascia or muscles.

Now with the point of a knife carefully puncture the abdominal wall just below the ensiform cartilage, making an opening of sufficient size to admit the first and second

* Virchow. "Post Mortem Examination.

fingers of the left hand. With these fingers elevate the abdominal wall, pass the section knife between the fingers with its cutting edge up, and moving both hands towards the symphisis, open the abdomen along its entire length. By proceeding in this way one avoids the danger of injuring the internal organs which exists if the body is opened by a second cut made as was the first.

In making the puncture below the ensiform cartilage notice whether there is an escape of gas, and whether or not, as the incision lengthens the intestines bulge up through the cut. If they do it indicates the presence of a considerable amount of contained gas.

XII. Should it be desired to make a bacteriological investigation of the peritoneum, or of the abdominal contents, tubes should be inoculated at this juncture, before the membranes or the organs become contaminated by further handling. After having inoculated such tubes as it is desired, the abdominal walls should be carefully drawn apart and a general inspection of the cavity made to note:—

1. Whether the organs occupy their normal position.
2. Whether there are any abnormal contents, such as tumors, fluids etc.
3. The general condition, color, etc. of the mesentery, peritoneum, etc.

No organs are to be removed at this time, because by so doing it is impossible to avoid severing the large blood vessels, such as enter the liver, spleen, etc., thereby allowing the blood to escape from the main veinous trunks, from the vena cava and thus from the right side of the heart, so that upon subsequently examining this organ, we do not find the conditions as they existed at the time of death, but possibly just the opposite.*

XIII. After having made the above observations the operator next

* Virchow, "Post mortem Examinations," page 78.

notes the position of the diaphragm on the two sides.* This is done by passing the right hand into the abdomen, over the surface of the liver on the right side and over the liver and spleen on the left till the highest point of the diaphragm is reached, with the left hand he notes upon the outside of the body, the rib, or intercostal space to which the diaphragm extends.

In the great majority of cases this can not be determined if the thorax is opened before the abdomen, or if after opening the latter we neglect to note the position of the diaphragm before opening the former, or before removing any of the abdominal organs, since in either case we can not avoid cutting through a portion of the anterior attachments of the diaphragm. It is in new-born children that it is especially important to ascertain carefully the position of the diaphragm since it is frequently of moment to be able to state positively whether or not the child has breathed and not only this, but but the extent to which the act has been carried.

## XIV. Opening the Thorax:-

Before proceeding to a closer examination of the abdominal contents we now pass to the opening of the thorax and general examination of its contents.° The reason for this apparently unsystematic proceeding has already been explained in a previous paragraph. (¶ XII, last half.)

Drawing the edge of the incision already made into the tissues of the chest, as far to the sides as possible, dissect away the muscles of the chest from their attachments to the ribs as far back on either side as can be conveniently done, disclosing the sternum, the costal cartilages and the ribs. Sever also the attachments of the abdominal muscles to the margins of the lower ribs.

---

* Normally the vault of the diaphragm reaches the fifth rib on the right side and fourth rib on the left side

° The only deviation from this rule is in cases of poisoning where the whole interest in the examination centres in the stomach and intestines.

Exchanging now the section knife for the stronger cartilage knife provided to remove the sternum in the following way, using considerable care not to injure the underlying organs by allowing the point of the knife to penetrate too far. Holding the knife firmly with its cutting edge up, sever the costal cartilages as close to their articulation with the ribs as possible, slanting the knife so as to give the cut surface of cartilages a beveled edge.

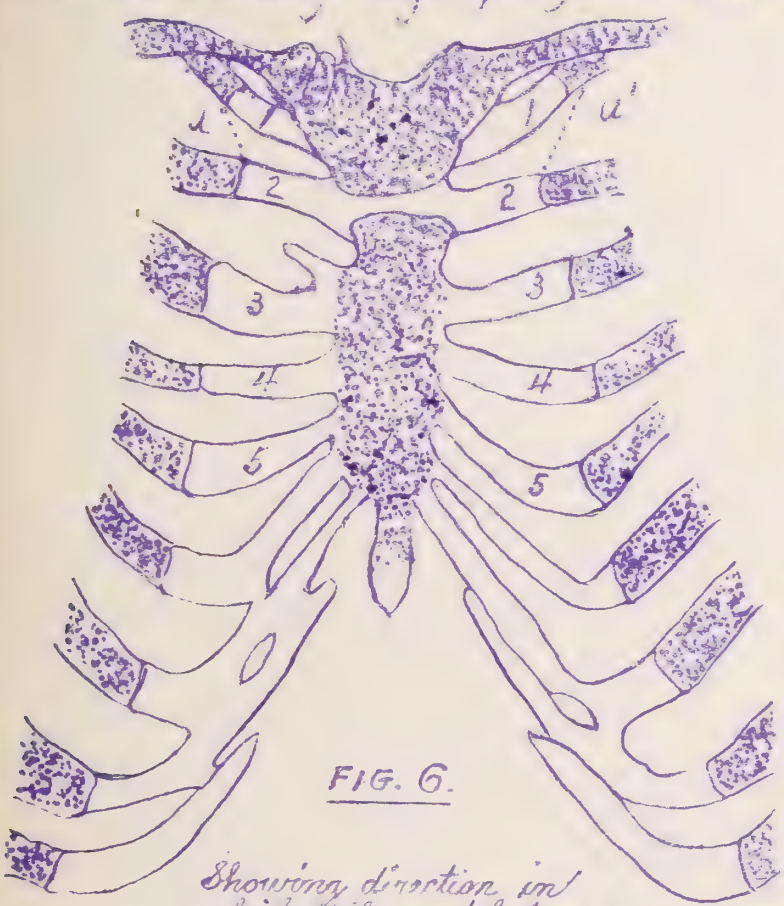


FIG. 6.

Showing direction in which knife must be turned to cut the cartilage of the first rib. See dotted lines a & a'

Ordinarily no difficulty is experienced with this procedure until the first rib is reached and here the cartilage can be easily cut if, instead of continuing the incision in the same line, the knife is slanted outward at an angle of about 30° (Fig. 6.)

Taking again the section knife direct the assistant to grasp the lower end of the sternum and cut cartilages, and gently raise the same while you dissect away the anterior attachments of the diaphragm, being careful not to enter the pericardial sac which lies closer to the under side of the sternum. As the tissues are severed from their attachment to the sternum care must be taken not to cut into any of the larger blood vessels especially the internal mammary, the internal jugular and the innominate, for by so doing fluid, or coagulated blood would escape and sooner or later find its way into one or both pleural cavities, in which event it would be obviously

impossible upon examination to state whether or not, in case these contained fluid, it had been bloody previous to opening the thorax, and it is necessary to point out how important it is in helping to form an opinion of a case, to be able to demonstrate positively the presence or absence of any foreign substance in these cavities

The sternum is finally completely removed by passing the knife through the sterno-clavicular articulations, which procedure is facilitated by directing the assistant to draw it strongly to the side opposite to the one which is being disarticulated, in this way widening somewhat the space through which the knife has to pass. After separating the articular surfaces the knife is directed outwards and slightly upwards, parallel to the clavicle, so as to cut thro' the rhomboid ligament.

In cases where, due to old age or disease, the costal cartilages have become calcified, it is necessary, to use the bone forceps, or the saw in cutting them.

Having now opened both the chief body cavities, it only remains to note the following points relative to the contents of the thorax before proceeding to remove and further examine the various organs.

1. Their relative position.
   A. Lungs
   B. Pericardial sac, also its shape and size.
2. The presence or absence of any foreign body
   A. Tumors
   B. Fluid, pleuritic or pericardial, and the character of the same.
3. The color of the exposed parts, especially of the pleura and pericardium, and the presence or absence of adhesions between these membranes and the organs which they surround.

# Chapter IV.

## Removal and Opening of the Heart.

XV. Up to this point we have considered only the general observations that are to be made upon the surface of the body under examination, the method of opening the same preparatory to the removal of the organs, and the chief points to be noted in connection with the contents of the chest and abdomen before such removal. Before taking up the subject of the detailed examination of the organs of the body let us see how these are most readily and systematically removed, for as we have already seen (Par. X and XII) there should be some system adopted whereby the removal of one organ does not interfere with observations that have to be subsequently made on other organs or parts. The necessity for such an order was pointed out in the case of the abdomen, where it was stated that none of the abdominal organs were to be removed before the chest had been opened and the heart examined. since such removal would necessitate the severance of several of the larger abdominal vessels whereby blood would be allowed to escape from the right side of the heart, leading to erroneous conclusions when that organ was afterward examined. The same thing holds true also of the remaining thoracic organs, the lungs, and while at first sight it would appear most natural to remove and examine these organs first, we see that this can not be done, and our examination therefore begins perforce with the heart itself which before removal should be partly examined in situ.

After we have opened the pericardium and determined its condition as mentioned in Par. XIV., we ascertain the following facts about the heart:

1. Its Position
2. " Size
3. " Color
4. Its Shape
5. " Consistency
6. The amount of blood in superficial Coronary vessels
7. The amount of fat about the organ.

The organ is now removed by cutting the aorta, pulmonary artery, and veins, and the vena cava, all of which have been previously ligated. After removal it is opened for examination. The chief points to be determined by an autopsy the heart are (1) the quantity and condition of the blood in its various cavities; and (2) the condition of the auriculo-ventricular orifices.

"The determination of the quantity and quality of the blood contained in the different parts of the heart is of decisive importance for ascertaining the kind of death. It is in the highest degree probable, even if not absolutely certain that two of the most important kinds of death, that from asphyxia and that from paralysis of the heart occur in consequence of the overfilling, in the first case of the right ventricle, and in the second, of the left." *

In order to secure the blood within the heart for measurement it is necessary to make four incisions, one into each cavity, and since we wish also to learn the exact condition of the various valves which close them, the general practice of those who we may call "primary incisions" is practically determined for us. Since they must be so situated as not to injure any of the valves at the base of the heart, their borders are those of the papillary muscles.

The following incisions are therefore made:—

(1.) Right Ventricle.— Begin close to the base of the right border. Pass the knife into the interior of the ventricle and continue it out nearly to the apex, being careful not to cut into the septum. (Fig. 7.)

* Virchow.— "Post-Mortem Examinations." P. 47.

(2.) Right Auricle. – Begin half way between the entrance of the venae cavae and end just in front of the base.
(3.) Left Ventricle. – Begin close to the base to the left of the mitral valve and continue almost to the apex. (Fig. 7.)
(4) Left Auricle. – Begin at the left pulmonary vein and continue to just in front of the base at a point usually indicated by a very prominent coronary vein.

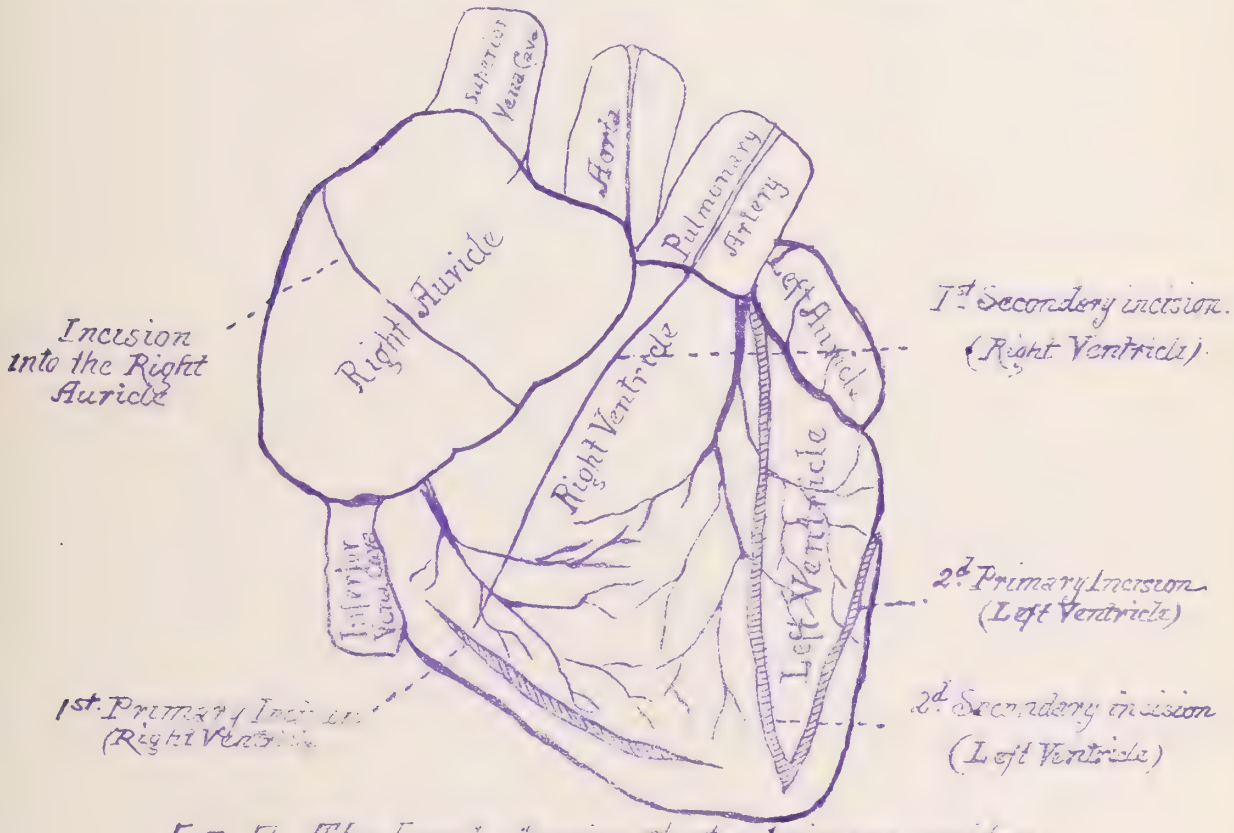


FIG. 7. The heart showing the two primary and two Secondary incisions into the Ventricles and the incision into the right auricle.

These cuts should for convenience, be called the "primary or p... incisions" for each cavity to distinguish them from others that are to be made later into the two ventricles. After making these carefully remove the blood from each chamber of the heart, determine its quality, whether fluid or clotted and measure it, after which measure the size of the mitral and

tricuspid orifices with the graduated cone, being careful not to remove from the valve segments any vegetations which may be present by rough handling.

The "first act" in the examination of the heart is now finished, and the "second act" consists in making the following observations.

(1.) Examine the cut openings of the aorta and pulmonary artery, to determine their size and the thickness of their walls.

(2.) Examine the closing power of the aortic valves by pouring water into the aorta while the heart is suspended.

(3.) Do the same with the pulmonary valves, in both cases being sure that no coagula attached to the valve segments prevent their closing.

The "third act" in examining the heart begins now with the opening of the two ventricles by what we may term the "secondary incisions," which are made in the following way:

(1.) Right Ventricle.- Begin at the cut end of the pulmonary artery and continue to the end of the primary incision already made, near the base. This incision should be made with a pair of scissors, care being taken not to injure the anterior papillary muscle of the tricuspid valve. (Fig. 7).

(2.) Left Ventricle.- Begin at the cut end of the aorta and continue close to the septum ventriculorum to the end of the primary incision already made near the apex. Care must be taken not to injure the base of the mitral valve on the left side, and not to cut into the pulmonary valves on the right. Carry the incision midway between the pulmonary orifice and the left auricle. (Fig. )

In concluding the examination of the heart we have to make the following observations:

(1.) The condition of all the valve segments
(2.) " measurements of the pulmonary and aortic valves.
(3.) " thickness of the right and left ventricular walls.
(4.) " depth and breadth of the ventricular cavities.
(5.) " condition of the endocardium
(6.) " condition of the coronary vessels
(7.) " condition of the intima of the aorta and pulmonary artery.
(8.) " presence of blood clots and their character, whether anti- or post-mortem. (For differences between these two forms of clots, see Lecture Notes, chapter on Thrombosis.)

All of the observations above mentioned should be carefully recorded.

## Chapter V.

## Removal of the Remaining Organs of the Thorax & Abdomen.

### Removal of the Lungs.

XVI. Usually the removal of the lungs is a simple matter and presents few difficulties. Occasionally however these organs are found to be more or less firmly adherent to the chest walls because of previous inflammation of their pleural coverings which has led to the formation of more or less firm fibrous adhesions. When this condition of things is found it is frequently difficult to remove the lungs without tearing to them & to the pleural membrane that covers them. When the adhesions are recent and fibrinous in character (the result of pleurisy) they are easily broken, but when such inflammation has led to the development of bands of fibrous tissue, the adhesions are sometimes extremely tough, and the pleura so much thickened that it has lost all semblance to the delicate membrane that normally covers the lungs. In this latter case the bands of fibrous tissue may

have to be cut in order to remove the lungs without injury, as any attempt to break them by force would result in tearing the pulmonary tissue. These dense adhesions and great thickening of the pleura are most frequently found at the apices, and in cases of tubercular disease of the lungs. In the removal of such an adherent lung it is frequently necessary for the operator to stand temporarily on the left side of the body.

After completely separating all adhesions the lungs are removed by cutting across the large bronchi and vessels at the root of each, avoiding any injury to the œsophagus and aorta. Some operators remove the lungs, œsophagus and aorta in one piece by cutting across these structures just below the clavicles, inserting the index finger of the left hand into the trachea, and while making traction, dissecting them away from the spinal column. After removal in this way the various structures are separated before examination.

## Removal of the Intestines.

XVII. In emptying the abdomen it is best to begin with the intestines, for after their removal all the other organs are more easily seen.

The omentum is the first object met with on attempting to remove the intestines, it is usually spread out over the abdominal contents extending almost to the symphasis pubis. It may however be found rolled up just below the stomach. Turn the omentum back over the chest and then proceed as follows:-

(1.) Find the duodenum below the point of entrance of the common bile duct. Apply a double ligature and cut the intestine across between these

(2) With the aid of an assistant holding the small intestine, "strip" it of its mesentery by cutting the same away close to its attachment to the intestines being

careful not to injure the gut.

(3.) When the coecum is reached draw it well toward the middle line of the abdomen. Separate the peritoneum and fibrous tissue on the right side, and pull up on the ascending colon cutting its attachments also. Toward the upper end of this portion of the intestines care must be taken not to cut into the portion of the duodenum left attached to the stomach.

(4.) Turn down the omentum and free it from its attachments to the transverse colon and stomach.

(5.) Draw the transverse colon well toward the middle line bringing the splenic flexure into view.

(6.) Free this portion of the intestine from surrounding tissue and follow it down, past the sigmoid flexure to the rectum.

(7.) "Milk" the contents of the rectum backward into the sigmoid flexure, ligate the rectum with a double ligature, cut it across and remove the intestines which are now entirely free.

## Removal of the Stomach, Duodenum & Pancreas.

XVIII. Before removing the stomach and duodenum, the latter should be opened, the opening of the common bile duct found, and if deemed necessary this passage should be explored as far back as the gall-bladder.* One may determine whether these passages are free by simply pressing on the gall bladder strongly when if no obstruction exists bile will be forced into the duodenum. This should always be done.

In order to remove the stomach it is only necessary to free the oesophagus from its attachments to the diaphragm, sever the common bile duct and free the organ from all surrounding tissue. It may now be lifted out with the duodenum and pancreas attached.

*In cases of poisoning a careful examination of these ducts can not be made.

## Removal of the Spleen.

XIX. This organ is found just below the diaphragm on the left side of the abdomen. It can be easily removed by drawing it carefully down with the left hand while with the right it is freed by a few cuts from any adhesions to the diaphragm or surrounding parts, its attachment to the tail of the pancreas having been already cut in removing that organ. It is often necessary to handle the spleen with great care as in a variety of morbid conditions it becomes so soft and friable that it is easily torn.

## Removal of the Liver.

XX. Before removing the liver the adrenal bodies should be taken out as there is danger of removing a portion of the right one with the liver to which it lies in close proximity. While the assistant raises the liver cut all its ligaments, break any adhesions that may exist between the right lobe and the diaphragm, where they are not infrequently found, after which the organ can be lifted out.

## Removal of the Kidneys

XXI. Before removing the kidneys, the last organs left in the abdominal cavity, find the ureters and trace their course from the bladder to the kidney pelves, noting any twists, kinks, dilations or strictures that may be present. To remove the kidney simply "shell" it out of the fat and connective tissue that surrounds it cut the ureter and renal vessels at the hilum.

## Removal of the Testes.

XXII. The testes should be removed before the pelvic viscera since by pursuing this order the normal relation of the parts is not disturbed. First find the vas deferens as it passes out

of the true pelvis and follow it to the internal ring. Should this structure not be easily found, as is the case where the pelvic contents have been already removed, find the spermatic veins which lie just beneath the peritoneum, and follow them to the internal ring. Here pick up the spermatic cord and pulling gently upon it draw the testicle into the abdominal cavity. Should there be difficulty in getting it through the rings these may be enlarged with the knife.

Possibly a more rapid, but less satisfactory way of removing the testicle is to push it out of the scrotum into the canal and so through into the abdomen, freeing it of whatever coverings of the canal hinder its progress. Usually on the removal of the testes the scrotum is seen to be invaginated. This is due to the fact that the tunica vaginalis is attached by connective tissue to the bottom of the scrotum.

# Chapter VII.

## Removal of the Male Pelvic Viscera.

## Removal of the Female Pelvic Viscera.

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XXII. By the male pelvic viscera we understand the bladder, prostate gland, rectum and urethra. The removal of these organs usually presents considerable difficulty, chiefly on account of the limited space in which one has to work. The following steps are to be taken:

(1.) Pull the bladder upward with the left hand and at the same time cut it away from the anterior abdominal wall, and from the back of the pubic arch, dissecting under the arch somewhat so as to free the prostate gland.

(2.) Do not sever the urethra at this point, but free the bladder from all connections to the sides of the true pelvis.

(3.) Draw the rectum toward the bladder and cut it away from the mesorectum.
(4.) Cut the rectum away close to the anus
(5.) Now lift up the pelvic viscera; pass the knife behind the pubic arch; cut across the urethra and having thus severed all attachments, remove the various organs.

XXIV. It sometimes happens that one desires to remove the entire genito-urinary tract in continuity, and in this case the method of procedure is entirely different from that just given for the removal of the pelvic organs alone.

One of the most frequent reasons for the removal of the entire genito-urinary tract in continuity is to enable the operator to follow up the changes that have been produced in the various portions of the tract by a long standing urethral stricture. This condition of the urethra is not infrequently accompanied by serious secondary changes in the bladder ureters and kidneys. Pressure on the urethra by an hypertrophied prostate gland may also lead to secondary changes in the other organs. In the removal of the various organs in continuity the following steps are to be taken:

(1.) Free the kidneys from all attachments.
(2.) Free the ureters in like manner from the kidneys to the bladder.
(3) Place the kidneys with ureters attached one on each side of the body, while the rectum, and bladder are freed as just described in section XXIII.
(4). Continue the first incision made in opening the body (Sec. XI.) over the symphisis and along the dorsum of the penis.
(5.) Separate the skin from the penis.
(6.) Cut across the penis just back of the corona, leaving the glans attached to the skin.

(7.) Dissect the penis away from the under side of the pubic arch and divide the crura of the penis close to their attachments to the rami of the ischia.

(8.) Push the penis, when freed, through the opening just made, into the pelvis.

(9.) Draw up the penis and bladder and free the prostate from its connections.

The kidneys ureters, bladder, rectum, prostate gland & penis can now be lifted out.

## Removal of the Female Pelvic Viscera.

**XXV.** In the female the pelvic contents consist of the ovaries, tubes and ligaments, uterus, vagina, bladder, rectum and urethra. These organs are removed in the following manner:

(1.) Cut away the bladder from the pubes and rami of the pelvis as described for the removal of this organ in the male. (Section XXIII.)

(2.) Free the rectum from its attachments to the sacrum as described in section XXIII for the male. This leaves the entire viscera attached to the floor of the pelvis only by the orifices of the rectum, vagina and urethra.

(3.) Unless there is reason, which there rarely is, for wishing to remove the labia minora with the vagina, it is only necessary now to cut away the rectum, vagina and urethra close to their orifices.

(4.) Sever the attachments of the broad ligaments on either side and the uterus, ovaries and tubes may be removed with the other pelvic organs.

# Chapter. VI. (a)

## Removal of the Brain & Spinal Cord.

XXVI.

The removal of the organs of the central nervous system, the brain and spinal cord, presents difficulties not encountered in the removal of the other organs for the double reason that they are very easily damaged because of their delicate structure and are in additions encased in thick walled bony chambers which it requires no inconsiderable force to enter, the use of which is only too apt to injure the contained structures. As difficult as the operation is it frequently has to be performed, though as a rule it is not necessary to remove the brain and spinal cord unless the patient showed symptoms of organic nervous disease pointing to lesions in these organs, or died from obscure causes.

The brain is removed in the following manner:

XXVII.

Opening of the Skull.

Slightly elevate the head by placing a block of wood beneath the neck. This block should be about 3-4 in high. If the body is that of a woman carefully unbraid the hair, and parting it from ear to ear and make a transverse incision from one mastoid process to the other. Draw the scalp forward, turning it inside out, so far as to disclose the superciliary ridges, and backward in the same way beyond the occipital protuberence.

The skull cap is now sawn through, the cut being started just above the superciliary ridges, high enough to pass above the orbital plate of the frontal bone and continued backward and downward on each side to a point just above the base of the mastoid process.

Turning the head first on one side and then on the other, the ends of these incisions just made above the

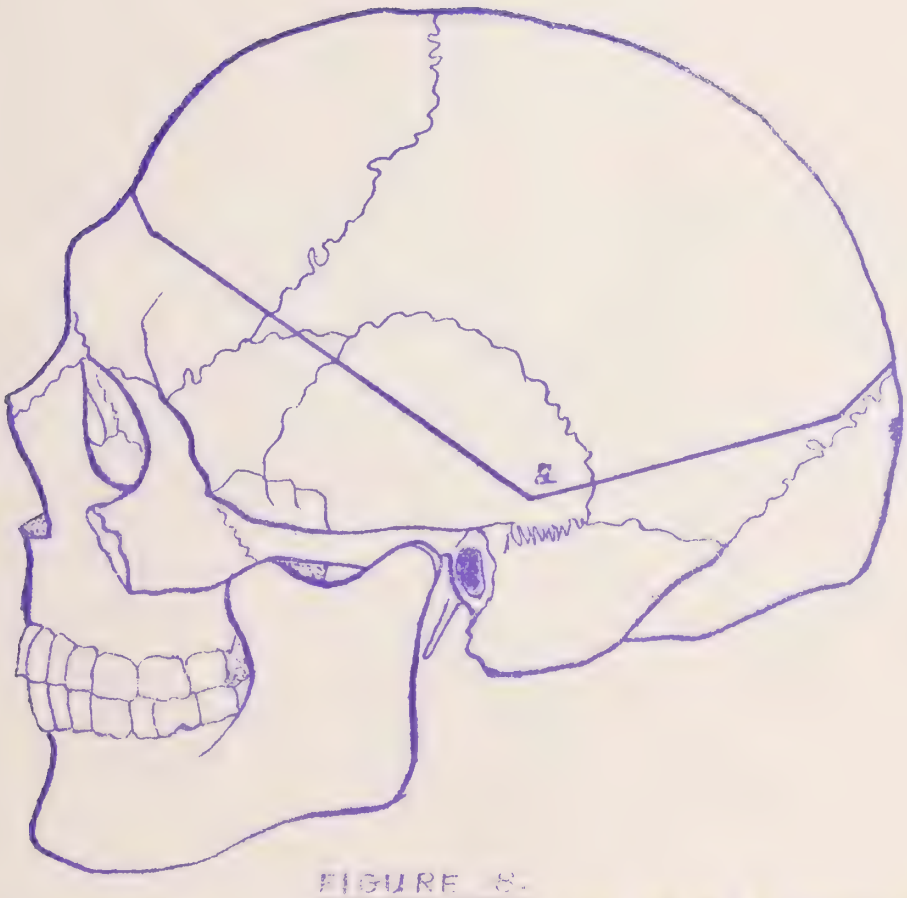


FIGURE 8.

Showing the incisions made in the skull for removing the skull-cap. The lateral incisions from an angle at a which prevents the cap from slipping when replaced.

mastoid process are joined by two saw cuts made on a backward and upward direction, so that they meet at the occipital protuberence. The result of these second incisions is the production of an obtuse angle upon each side of the skull with the apex pointed downwards and located just above the base of the mastoid process. Into these the skull-cap fits and when replaced after the autopsy it is thereby prevented from slipping forward and producing unsightly deformities of the head.

In making the cuts with the saw the bone only should be sawn through, the dura mater left intact. After the completion of the above mentioned incisions the skull should be prized up by the hand, hook, or by the handle of the post-mortem hammer. The use of the hammer and chisel for this purpose should be avoided if possible especially in medico-legal cases, for there is always a liability of producing fractures by their use. If as sometimes happens, in children and especially in the aged, the dura mater should be found adherent to the inner plate of the skull the skull cap should be slightly raised and the dura mater divided around the edges of the saw cut with a pair of scissors.

## XXVIII.

### Removal of the Brain.

Having carefully removed the skull-cap, and where necessary, the dura mater from the brain, introduce the first finger of the left hand beneath the frontal lobes and lift the brain up from the base of the skull. With a medium sized scalpel divide all the vessels and nerves as they appear at the base of the organ as closely to the bone, or dura mater as possible. When this is done raise the brain as high as possible and passing as small knife far down each side of the medulla [illegible] the tentorium

atlas and then cut the cord across. The brain is now free and can be lifted out.

## XXIX.

### Opening of the Spinal column.

Place the body on its ventral surface and make an incision over the median line from the occipital protuberence to the upper border of the sacrum. Carefully remove all the muscles from the spines and laminae by reflecting them to either side. After having done this and before proceeding to open the spinal canal carefully examine for any signs of fracture or dislocation, and note any peculiarities of the vertebrae that may be present. At times dislocations may be present which are not readily recognised from behind and it is therefore always wise to examine the bodies of the vertebrae from in front, after having removed the organs of the thorax and abdomen.

Begin to enter the spinal canal by sawing through the laminae on either side, being careful to direct the saw downward and inward, for otherwise, if the saw is directed only downward it may pass entirely without the canal. Sometimes, after having made an entrance at one point it may be more rapidly opened by severing the remaining laminae with a bone forceps. These are usually necessary at any rate to cut through the arches of the Atlas and Axis. Having severed all the laminae it is only necessary to remove the ligamente attached to them by drawing a spinal chisel along the line of incision on each side, when the arches may be lifted up, disclosing the spinal cord below covered with its dura mater.

## XXX

### Removal of the Spinal Cord.

To remove the cord take the following steps:

(1) Carefully pass a thin bladed knife along each side of

it, outside the dura mater, severing the anterior and posterior roots.

(2.) Divide the cauda equina.

(3.) Lift up the lower end of the cord and see that it is entirely free up to the foramen magnum. In doing this the greatest care must be taken not to make traction on it. It is best in dividing the cord as stated in (2) above to leave sufficient of the cauda equina to grasp readily in the forceps.

(4) Cut through the dura mater at the edge of the foramen magnum to which it is attached.

(5.) Having already cut the upper end of the cord in removing the brain now carefully draw it out.

(6.) Place the cord on some smooth surface and with a small pair of scissors open the dura mater in the anterior median line.

# SECOND PART.

## Chapter VII.

### The general Examination of the Organs that have been Removed.

XXXI.

Up to this time we have considered practically only the technique to be employed in conducting an autopsy; that is we have studied the steps that have to be taken in removing the different organs from the body, only incidentally referring to a few of the chief points that must be observed and recorded as we proceeded. The remainder of these notes will deal with the detailed examination of the organs and structures that have been removed, or which have been disclosed for examination in opening the various cavities of the body. The chief changes that one is likely to meet with will

be mentioned and their significance explained. By far the greater number of changes met with have already been described in the body of this annual under the several paragraphs devoted to a description of the "pathological anatomy" of the morbid changes found in the different organs, these will therefore be omitted in what follows, reference being made to the preceding pages upon which they have already been described.

It is of prime importance that a physician when performing an autopsy should be able to correctly translate the changes with which he meets. This really is the essential requirement; for of what value would be the most refined technique, if after having properly removed an organ, one was unable to say whether or not it was normal, and if found to be altered, in what the alterations consisted and what it signified?

In the second chapter we noted the chief points to be observed in making an examination of the exterior of the body without going into details, the changes likely to be observed, except in comparatively rare instances, being easily understood. The size of this little manual will not permit of a discussion of those changes that are the least likely to occur, as important as they may be. While therefore the smallest details must be observed and recorded, space will not permit us to enter into an explanation of all that may be found.

In describing in detail the internal organs and structures of the body, we will begin with

## XXXII.

## The Peritoneum.

After opening the abdomen as directed in paragraph XI, and making the general observations there mentioned, carefully examine the delicate membrane that lines the abdominal cavity and covers its organs, the peritoneum,

it should be everywhere smooth, glistening and pale in color.

Any of the following appearances may be observed and denote the presence of different morbid processes. The chief changes only are given.

XXXIII.

Simple Acute Peritonitis.

(1.) The peritoneum has lost its glistening appearance either in places or everywhere.

(2.) Instead of being smooth, it is, in places, or generally, slightly roughened, granular in appearance, and may even be covered with a definite film of fibrin that can be easily scraped off.

(3.) The intestinal coils will sometimes be found glued together by a sero-fibrinous exudate.

(4.) Close observation reveals the engorgement of the small vessels of the peritoneum which in many cases is so pronounced as to give the membrane a reddish, or even an angry red color.

XXXIV.

Purulent Peritonitis.

This condition presents all the changes above mentioned to which may be added the presence of pus in the abdominal cavity, bathing the peritoneum and usually the intestinal coils. This form of inflammation is always due to some pyogenic microorganism, the ones most commonly found on bacteriological examination being the Streptococcus pyogenes, or the bacillus coli communis. In consistency, the intestines seem to be more flabby than normal.

XXXV.

Chronic Peritonitis.

(1.) The peritoneum is found to be thickened in places, or generally is opaque, having lost its normal lustre.

(2.) It is of a greyish color.

(3.) Not infrequently there is present considerable fluid in the abdominal cavity. All signs of an acute inflammatory process, such as those given in paragraph XXXIII, are absent. There is no engorgment and no exudate of fibrin as above mentioned.

XXXVI.

*Tubercular Peritonitis.*

In this condition the peritoneum and mesentery are found to be studded with small greyish nodules, varying in size from that of a pea to that of a millet seed. They are larger than the nodules usually found in cases of acute miliary tuberculosis and are less translucent. Where they are as large as small peas they are yellowish in color. These nodules are tubercles.

XXXVII.

*Malignant Disease.*

Closely simulating the above appearances are the multiple metastatic nodules of certain forms of malignant tumors which, however are rare in comparison with tuberculosis of the peritoneum. It is usually impossible to distinguish between them without the aid of the microscope. The presence of the primary tumor and the absence of tuberculosis in other parts helps one to make the diagnosis.

XXXVIII.

*Old Hemorrhages.*

These are indicated by the presence of scattered patches of a slate color.

XXXIX.

*Old Adhesions.*

These may be found binding one or more coils of the intestine together, or to the parietal peritoneum. In either case they indicate the fact that at some previous

time the peritoneum has been inflamed, resulting in the organization of the exudate and the production of bands if fibrous tissue. Sometimes this takes place to a marked degree about the hepatic vein as it leaves the liver to empty into the vena cava, and the gradual contraction of this connective tissue leads to a narrowing of the hepatic vein and the consequent production of a passive congestion of the liver. This condition is sometimes spoken of as proliferative peritonitis.

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## The Intestines.

**XL.**

### Perforation due to Ulcers, to Appendicitis and to Strangulation.

In cases where an acute peritonitis has been found the entire length of the entestine should be carefully examined as well as the stomach for any possible perforation through which feces may possibly have escaped and set up the peritoneal inflammation. Perforations are usually due to ulceration, the ulcers being chiefly due to typhoid fever and being located in Peyers patches in the lower portion of the ileum and about the coecum. Tubercular ulcers rarely perforate. (For the explanation of this fact see the chapter on Tubercular ulcers of the intestine in the body of the manual, page 114) Perforations may also be due to gangrene of the gut resulting from strangulation or to the rupture of an appendicoecal accumulation of pus. These conditions should be carefully looked for.

The intestines should also be examined for the following conditions:

(1.) Contractions.
(2.) Dilitations.
(3.) Strictures.
(4.) Invagination { True / False
(5.) Volvulus
(6.) Strangulation
(7.) Fecal impaction
(8.) Tumors.

XLI.

Contractions.

Contraction of the large intestine is quite commonly found, and in some cases follows suddenly extreme dilitation. The condition is unimportant and is probably to be classed as a post-mortem or "agonal" phenomenon.

XLII.

Dilitations.

These are usually found immediately above some obstruction in the gut, are produced mechanically. In some cases especially in children an extreme dilitation of the large intestine is found. An interesting case of this kind has been described by Osler When the dilitation is great the walls of the intestine may be quite thin and anaemic.

XLIII

Invagination.

Two forms of invagination are found and must be distinguished from one another. These are the true invaginations which have occurred during life, and invaginations occurring post-mortem, or as "agonal" phenomena. True invaginations always takes place from above downward. The peritoneal surfaces of the entering and returning guts are frequently stuck together by a fibrinous exudate. The mesesentary about this portion of the intestine is inflamed and thickened.

Post-mortem invagination on the other hand is free from all signs of inflammation, is readily reduced and may occur either from above downwards, or the reverse. It most frequently is found at the junction of the ileum and caecum.

XLIV

*Volvulus or Twist.*

This condition is most often found in the large intestine. The parts are discolored and sometimes gangrenous, which is due to the cutting off of the circulation by the closure of the vessels in the knotted portion.

XLV.

*Strangulation*

This is not common. Occasionally a hernia becomes irreduceable and the gut strangulated at one of the rings. Under these circumstances gangrene quickly ensues, unless the trouble is remedied by operation. Rarely the intestine becomes kinked as it were, and the circulation being in this way cut off the part becomes gangrenous. Very rarely also, contracting bands of adhesions formed by some previous inflammatory process, may cause a strangulation

XLVI.

*Tumors*

Tumors of the intestinal walls are chiefly found in the large gut. In the majority of cases they are malignant in character and should be carefully examined after removal. At times the tumor is of considerable size. The walls of the intestines are generally greatly thickened and its lumen correspondingly reduced in size. Above this constriction the gut will be found to be dilated

XLVII.

*Opening of the Intestines*

The method of removing the intestines has already been given. It now only remaines to open it and carefully examine its mucous membrane. Usually it is advisible to leave this till the last as it is at best

a dirty piece of work. Where there is a sink of considerable size, with running water at hand it may be possible to flush the entire length of the intestinal canal by attaching one end to the faucet and allowing the water to flow through it. In the majority of cases this is impossible and the organ must be washed as it is opened.

For opening the intestine we use what is called an enterotome, a large pair of scissors, one blade of which is longer than the other and rounded or blunt (Fig. 3 page 4). Introduce the blade with the blunt end into the canal and draw the intestine over it, making the incision along the line of the mesenteric attachment in order not to cut into Peyers patches which are situated opposite to this border, and which in certain morbid conditions are the the seat of important lesions. On account of its size the vermiform appendix must be opened with a smaller pair of scissors.

XLVIII.

Normal Mucous Membrane.

In the normal intestine the mucous membrane is thrown into folds known as the "Valvulae Conniventes"; these are most numerous and of larger size in the upper portion if the small intestine, diminishing in size and number as we approach the ileo-cecal valve, finally vanishing altogether before this part is reached. These folds may be stained with feces and sometimes with bile.

XLIX.

Peyer's Patches.

In the lower portion of the ileum and lymphoid structures known as Peyer's Patches are found. Much smaller lymph glands, the solitary follicles are also

found between the larger patches, which are simply aggregations of the smaller glands. The Peyer's Patches are usually oval in shape, sometimes round, are slightly elevated above the general level of the mucous membrane, are slightly paler in color, and when oval, lie with their long axis in that of the intestine. These structures are quite prominent in children, and in adults, when diseased, while in advanced life they undergo partial atrophy and are in consequence much less easily seen. In young persons also smaller, solitary glandular bodies can be easily seen in the large intestine as slightly elevated, round bodies which diseased may become quite prominent. The following pathological changes are most frequently met with in the intestine:

L.

Oedema.

In cases of advanced nephritis, or in any condition associated with general oedema the mucous membrane as well as the other coats of the intestine are found to be swollen, pale and gelatinous in appearance.

LI

Congestion.

Injection of the small vessels of the mucous membrane is associated with any condition which cause a damming of the blood in the portal circulation. The most frequent causes of this are advanced cardiac valvular lesions, and that form cirrhosis of the liver in which the portal veins are constricted. (Atrophic Cirrhosis, see Chapter on Cirrhosis of the Liver, Page 20.) Where the congestion has been severe small hemorrhages may have occurred.

LII.

Inflammation, Acute Enteritis, Acute Colitis, Acute Entero Colitis, Acute Proctitis.

Inflammation of the intestine is not always easily discovered at autopsy. Usually in simple varieties one finds some congestion of the mucous membrane which at the same time is covered with a thick, sticky mucous exudate. In other cases there will be no appearance to point to an inflammatory process. Occurring in the small intestine, an inflammatory condition is known as "acute enteritis". If in the colon as "acute colitis". Occurring in both the small and large intestine it is spoken of as "acute entero-colitis". The lymphatic structures are ~~are~~ usually swollen, and in the colon small irregular ulcers of the mucous membrane may be found. When the inflammation is confined to the rectum it is spoken of as "acute proctitis."

LIII.

For the condition of the intestines in the following diseases see the various chapters on the intestine in the Manual:

(1.) Typhoid Fever. (Page 111.)
(2.) Tuberculosis ( " 114.)
(3.) Tropical Dysentery ( " 118.)

LIV.

Simple Acute Dysentery

Dysentery of a simple variety is a more severe inflammation of the colon than that described as Acute Colitis. The following changes are found:-

(1.) The mucous membrane is injected and swollen.
(2.) It is bathed with serous, or purulent fluid.
(3.) Hemorrhages into the mucous membrane and into the lumen of the intestine are of frequent occurrence.

(4) Ulcers of the mucous membrane, and of the solitary lymph glands occur. When these glands are not ulcerated they are ~~[illegible]~~ swollen and prominent, and larger than in cases of acute colitis.

## LV.

### Diptheretic Colitis.

In very severe forms of inflammation of the colon the exudate that is formed in diptheretic inflammation of the bowel. (See Lecture Notes for the definition of a diptheretic inflammation, Page 36) The surface of the intestinal mucous membrane is covered with a layer of fibrin that extends deeply into the submucous tissue at points where the mucous membrane itself has undergone necrosis. In attempting to remove this fibrinous exudate one tears away a portion of the underlying tissue. This condition is in reality a form of dysentery, the term diptheretic colitis being preferable to dysentery as it indicates both the location and the character of the inflammatory process.

## LVI.

### Chronic Dysentery.

The following changes are found in chronic dysentery.

(1.) Thickening of the intestinal walls, the mucous membrane being thrown into folds in a longitudinal direction.

(2.) The intestinal lumen is reduced in size.

(3.) The mucous membrane is absent in places as the result of ulceration.

(4.) Slate colored patches here and there indicate the position of previous hemorrhages into the mucous membrane.

## LVII.

### Other Intestinal Ulcers.

Aside from the conditions just described which are associated with intestinal ulcers ~~we~~ may have ulcers under the the following conditions:

(1.) Rarely in cases of Bright's Disease, associated with hemorrhages, and found especially in the caecum and ileum.
(2.) From the irritation and pressure produced by hard faecal masses.
(3.) In cases of chronic diarrhea small ulcers of the follicles are found.
(4.) In cases of intestinal obstruction round ulcers may be found above the point of obstruction, the so-called "distention ulcers."
(5.) In chronic pulmonary tuberculosis small ulcers of the solitary follicles may occur which are not of tubercular origin.
(6.) In cases of poisoning by bichlorides of mercury, small, round, or linear ulcerations of the mucous membrane of the caecum and colon are occasionally to be found.

## LVIII

### Polypi.

Occasionally single or multiple polyps of the intestine may be found. These occur chiefly in the colon. In conditions of general glandular enlargement the lymphoid structures in the intestine become very prominent.

# The Stomach.

## LIX.

### Opening the Stomach.

Opening the stomach with an enterotome along its greater curvature because:-

(1.) This gives a better view of its walls, and
(2.) Ulcers when present are most frequently found along the lesser curvature

Rugae when present show that the stomach was empty and contracted at the time of death, their absence indicating the opposite condition.

The contents of the stomach should be carefully examined for any abnormal constituents and in order to note the extent of digestion If desired for further chemical and microscopical examination they should be saved. If the case is one upon which the autopsy was done for medico-legal purposes, and poisoning is suspected, the vessel containing the stomach contents should be carefully sealed and labeled. If there is food in the stomach at the time of death it will be found accumulated at the cardiac end and in consequence, the presence of congested vessels of a blackish color, so frequently observed in this portion of the organ, is to be explained by the action of the acid contents of the stomach upon the blood.

## LX.

### Post-Mortem Digestion of the Mucosa:

Post-mortem digestion of the mucous membrane of the stomach not infrequently occurs and is chiefly found in the cardiac end also. It is distinguished from ulceration by the following:

(1.) It is extensive and diffuse.
(2.) The loss of substance is gradual.
(3.) There is no induration about its edges.
(4.) There is no signs of acute inflammation and no chronic inflammation of the peritoneal coat.

## LXI.

### Congestion.

This is denoted by the same signs as already given for congestion in the intestines. It is due to the same causes.

## LXII

### Sub-Acute Gastritis.

This condition is denoted by the following appearances:
(1.) Congestions of the small vessels in the mucosa associated with,
(2.) The presence of thick tenacious mucous. (See inflammation of the intestines. Page 34, paragraph LII.)

## LXIII

*Chronic Gastritis.*

In this condition the following changes are found:

(1.) Atrophy of the mucous membrane.

(2.) Thickening of the sub-mucous layer.

(3.) Pigmentation of the mucous membrane especially in the neighborhood of the pylorus.

## LXIV.

*Gastric Ulcer, Acute.*

This disease, most frequently observed in women between the ages of 18 and 25 presents the following appearance at autopsy.

(1.) The ulcer is most frequently located on the posterior wall of the stomach, in the lesser curvature, near the pylorus.

(2.) There is usually but one ulcer. Rarely the ulcers are multiple.

(3.) The ulcer has sharply punched-out edges.

(4.) These borders are not thickened.

(5.) The mucous membrane is more extensively involved than the sub-mucous coat.

(6.) There are signs of inflammation of the peritoneum over the seat of ulceration and, in some cases adhesions to surrounding parts.

## LXV.

*Gastric Ulcer, Chronic.*

Presents the following characteristics

(1.) The location is the same as in the acute variety.

(2.) There is usually one ulcer, but more often than in the acute variety, are found several.

(3.) The edges of the ulcer are indurated and swollen.

(4.) The floor of the ulcer is formed by the sub-mucous or muscular coats, or after gradual perforation has taken place, by some adherent organ as the pancreas, etc., the

escape of stomach contents being prevented by the gradual progress of the ulceration, allowing time for the inflammatory process set up in the peritoneal coat over the base of the ulcer to glue the organ so firmly to other parts that when actual perforation takes place no escape is possible.

(5.) Haemorrhage may have taken place into surrounding parts, leaving slate colored patches.

(6.) Erosion of some artery may have taken place in the progress of the disease, therefore examine for such a condition.

## LXVI.

*Pyloric Stricture.*

Not infrequently when the stomach has been the seat of ulceration the result will have been a gradual narrowing of the pyloric end, with considerable thickening of its walls. This condition must not be mistaken for malignant growth with which it can easily be confounded as this is so frequently found in the region of the pylorus. The pre-existing ulceration which has given rise to the stricture should be carefully sought for.

# Chapter VIII.

## Examination of the Lungs, Pleurae & Pericardium.

## LXVII.

*The Ribs, Sternum and Costal Cartilages.*

After having dissected away the skin and muscles from the thorax as already directed, the ribs, sternum and costal cartilages should be examined. Any of the following conditions may be found.

(1.) Calcification of the cartilages. This occurs normally in advanced life. It is said to occur frequently in the cartilage of the first rib in cases of pulmonary Tuberculosis.

(2.) At the junction of the cartilages with the ribs there may be found sometimes especially on the underside a marked beaded appearance. This is an indication of rickets and is found in children suffering with this disease.
(3.) Notice any bony growth of the ribs, sternum or clavicles.
(4.) Notice the shape of the sternum. It may be bulging and prominent or depressed forming the so-called "funnel breast."
(5.) Examine the underside of the rib and sternum for possible erosion due to aneurism of the aorta.
(6.) Examine for fractures, or result of old fractures of the ribs

## The Lungs.

LXVIII

Reasons for failure to Conclude.

Upon opening the thorax, the lungs if perfectly normal should collapse. It is comparatively rare however that they do so because one condition or another will prevent, the most frequent of which are the following:

(1.) Pleural adhesions.
(2.) Emphysema
(3.) Collections of mucous in the bronchi which cannot be expelled by the mere force of the elasticity of the lung tissue drawing the air out.
(4.) Consolidation of the lung such as is found in coal pneumonia, extensive broncho pneumonia, or advanced tuberculosis.
(5.) Extensive fibroid changes in the lung.

The chief external appearance of the body have been noted for discovering the pleura. It is only necessary here to mention the fact that the pleura frequently present scar-like depressions and puckerings due to contraction of bands of interstitial tissue in nearly every case indicating the presence of a chronic or of a healed tuberculosis at this point.

## LXIX.

*Color.*

In children the color of the lungs is pale pink, while in adults there is always found more or less pigmentation, the pigment being deposited in the lymphatics of the under layer of the pleura. (See, "Dust Pigmentation of the lung," page 63-64 of the Manual.) Where death has been accompanied by violent dyspnoea small sub-pleural hemorrhages will be found scattered here and there over the surface of the lung

## LXX.

*Section of the Lungs:*

The best method to pursue in opening the lungs is to first examine the larger bronchial tubes by slitting them up with a pair of scissors. It is advisable to first open the branch which descends near the posterior median border, as in thus proceeding but little of the lung tissue is injured. Following up its branches note any presence of mucous, pus, blood, or food particles. Note also the color and condition of the bronchial mucous membrane, which is occasionally much stained with blood. The appearance is post-mortem and of no moment.

## LXXI.

*Bronchitis.*

Injection of the vessels of the bronchial mucous membrane combined with the presence of a considerable amount of thick mucous indicates the fact that there has been a bronchitis in progress at the time of death

## LXXII.

*Bronchiectasis.*

Dilitations of the smaller, or even of the larger bronchial tubes may be found. This condition is known as "bronchiectasis," and occasionally large sacculations filled with a foeted muco-purulent material may be found. In some

cases the shape of these dilatations is globular, while again there may be dilatation of a tube for considerable distance leading to the formation of a cylindrical cavity.

The bronchi that pass deeply into the substance of the lung should not be opened unless there is good reason to suspect important changes, as it damages the lung too much for further examination by section.

The branches of the pulmonary artery should be examined for possible thrombosis, or the presence of emboli.

## LXXIII

### The Bronchial Glands.

The bronchial glands have been proven to be the starting point for general tuberculosis in so many cases that their examination should never be neglected. In children these glands, which are to be found at the root of the lungs, are especially prominent, as are all the lymphatics, while in adults they are smaller in comparison to the other organs and have changed from a pale pinkish color to being generally deeply pigmented. Several of these glands should be removed for future microscopic examination.

In most cases of pulmonary tuberculosis these glands are found to be involved and many of them are caseous. It is now believed that a certain percentage of cases of pulmonary tuberculosis have their starting point in tuberculosis of these glands.

## LXXIV.

A description of the following changes in the lungs will be found in the body of the manual. No mention of them will be made here.

(1.) Emphysema.
(2.) Atelectasis
(3.) Pulmonary Oedema
(4) Chronic Passive Congestion
(5.) Hemorrhagic Infarction
(6.) Dust Pigmentation.
(7.) Acute Pneumonia.
(8.) Broncho Pneumonia
(9.) Chronic Interstitial Pneumonia.
(10.) Pulmonary Tuberculosis.

## The Pleura.

The following are the chief conditions likely to be met with in examining the pleurae and pleural cavities.

### LXXV.

*Hydrothorax.*

This condition is characterised by an accumulation of fluid within the pleural cavities, which is serous in character, and is due either to passive congestion, the result of serious valvular lesions of the heart, or is found in conjunction with serous effusions in other localities in cases of advanced parenchymatous nephritis. The signs of inflammation are always lacking, and the condition is in the great majority of cases of bilateral.

### LXXVI.

*Adhesions.*

Adhesions between the pleura covering the lungs and the parietal pleura are mainly of two kinds, (1.) delicate bands of fibrin, the result of the coagulation of an inflammatory exudate that has been poured out over the pleural surfaces. These fibrinous bands often serve as a ground substance and possibly as food for developing connective tissue which springs up and develops into firm fibrous adhesions binding the lung firmly to the chest walls. This process of developing is sometimes spoken of as "organising", and the adhesions thus formed constitute the second variety found. At first these fibrous bands are very delicate and easily broken, but the older

they become the more they contract and the firmer they get, so that in time they distort the surface of the lung very much and are broken with difficulty on attempting to remove the lungs.

## LXXVII.

### Acute Pleurisy.

By this term is understood an acute inflammation of the pleural membranes which is characterised by the following appearances.

(1.) The surface of the pleura which is normally smooth and glistening has become dull and opaque.

(2.) It is seen to be slightly roughened and granular.

(3.) Not infrequently it is covered with a layer of fibrin which can be easily scraped off.

(4.) The small blood vessels of the membrane are found to be congested.

(5.) If the pleurisy has advanced to the stage of effusion there will be an accumulation of fluid in the pleural cavity of the affected side. In this fluid flakes of fibrin are generally found. Rarely the fluid instead of being serous, or sero-fibrinous in character is purulent, and the condition is then known as "empyema," or sometimes is spoken of as "pyo-thorax." More rarely still the fluid found in the chest cavity is bloody, which is either due to tuberculosis, or to some malignant growth.

## LXXVIII.

### Chronic Pleurisy.

This condition is usually localized and is most frequently found at the apices. It is characterized by:

(1.) The presence of firm fibrous adhesions.

(2.) Puckering and scarring of the surface of the lung.

(3.) Thickening of the pleura. This is sometimes very marked.

# The Pericardium.

## LXXIX.

### Normal Appearance.

Like the other serous membranes that we have examined the peritoneum and the pleura, the pericardium normally presents a glistening, smooth surface of a whitish color. When inflamed it presents the same appearances described already in speaking of inflammation of the pleura and peritoneum. That is glistening, smooth surface is converted into a dull opaque one that is decidedly granular and is not infrequently covered with fine filaments of fibrin that have stretched between the parietal pericardium and that covering the heart. On removing the heart these have been broken and portions clinging to the surface of the heart or to the parietal pericardium give it a shaggy look.

As in the case of acute pleurisy, there may or may not be a serous effusion into the pericardial sac.

## LXXX.

### Hydro-Pericardium.

In cases in which hydro-thorax has been found one is likely to find the same condition involving the pericardial sac, which in these cases is partly filled with a watery fluid. All signs of inflammation are wanting, the condition being part of a general dropsy in most cases. Not infrequently a small amount of such fluid is found in the pericardium in cases that have died of advanced phthisis.

## LXXXI.

### Blood and Blood Clot in the Pericardium.

Where blood or blood clot is found in the pericardium it is probable that there has been a rupture of an aneurism. If this has taken place suddenly the heart will stop rapidly if there has been an escape of ten

ounces while if the blood escaped slowly a much larger quantity can accumulate before death ensues

## LXXXII.

### Milk Spots.

Whitish spots most frequently seen on the visceral layer of the pericardium, and chiefly over the ventricles, and in hypertrophied hearts, are due to local thickening of the membrane and are known as "milk spots".

## LXXXIII.

### Chronic Pericarditis.

This condition, like chronic pleurisy, is chiefly characterized by the presence of numerous, rather firm fibrous adhesions that bind the two layers of the pericardium together, and by the absence of all signs of acute inflammation.

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# Chapter IX.

## The Heart.

## LXXIV.

### Contents of the heart cavities.

The method for opening and removing the heart has already been given. (Chapter IX, pp. 76-78.)

The first observations to be made are upon the character, or tendency, and distribution of the blood, or blood clots within the organ. The heart may have died in systole that is firmly contracted, in which case its action probably ceased before or with the cessation of respiration, and the cavities of the ventricles are empty in consequence of the firm contraction.

In cases on the other hand where respiration ceased before the heart it does in diastole, that is dilated and the right ventricle is always found to be full of blood, while

there is a lesser quantity in the other cavities. Where death was due to suffocation the right side of the heart is found to be distended with blood. Where death is due to paralysis of the heart the left ventricle is overfilled.

## LXXXV.

### Heart Clots anti- and post-mortem.

It is always of the greatest importance, when one finds clots of blood in the heart cavities, to be able to state whether or not they were formed before or after death; for in the former case they are of importance in explaining the cause of death, while in the latter their presence is of no especial import, blood naturally clotting in the heart cavities and larger vessels when the circulation ceases. For the points of difference between anti- and post-mortem clots see the Chapter on Thrombosis in the Lecture Notes.

## LXXXVI.

### The Valves.

Having thoroughly examined the contents of the heart our attention is next directed to the various valves of which there are four - the Aortic, Pulmonary, Mitral and Tricuspid. We have to determine the following points :-

(1.) The size of the orifices.
(2.) The condition of the valve segments.

## LXXXVII.

### Size.

In order to ascertain the size of the various orifices it is best to employ the graduated cone which is inserted into each of the openings in succession until it just fits. The diameter of the opening is denoted by the reading on the cone. (For the normal size of the various openings consult the table of measurements, etc., in the last chapter.

## LXXXVIII.

*Condition of the valve segments.*

The most important observations on the heart refer to the condition of the valvular segments. These in their normal condition are delicate, thin, membranes either arranged as cusps, as in the aortic and pulmonary valves, which by becoming filled with back flowing blood in the aorta, or pulmonary artery, fall together and so close the opening, or as in thin membranes one upon each side of the opening, hanging down into the cavity of the ventricle in such a way that when the blood is forced against them they come into accurate apposition, thus closing the orifice. This class of valve is found closing each auriculo-ventricular opening.

## LXXXIX.

*Valvular Lesions.*

Two chief forms of valvular lesions are met with: (1.) Those in which, due to some injury to the segments themselves, the opening is not fully closed when these segments come together, and (2.) those in which, due either to the contraction or to the enlargement of the opening, the segments do not accurately close the opening. In this latter form of valvular lesion the segments themselves are not at fault, but simply do not fit the opening that they were originally designed to close.

## XC.

*Testing the Competency of the Valves.*

In order to determine whether or not such changes in the size of the openings have occurred as will render them incompetent we have to measure them as already described and compare these measurements with the other dimensions of the heart in order to determine approximately whether or not they are relatively in proportion, to the rest of the

organ, and if possible to apply some mechanical test to further determine whether or not the opening is or is not actually closed when the segments come together. This is accomplished with comparative ease in the case of the aortic and pulmonary valves where it is only necessary to suspend the heart and pour water into the aorta and pulmonary artery, before the organ is opened, when if the closure is complete no water will escape into the heart. In the case of the auriculo-ventricular valves this method is not so accurate, and cannot of course be applied after the heart has been opened. Before the heart is opened we can pour water into the ventricles through the auricles and see whether the valve segments float up as the cavities become filled, into close and accurate apposition. Our greatest reliance however must be placed on the measurements first mentioned. Both of these methods are at best far from giving us any accurate knowledge because of the fact that the auriculo-ventricular valves depends very largely on the muscular contraction of the heart which after death can not be reckoned with.

Lesions of the valves such as just described are mostly found in hearts that have undergone hypertrophy and secondary dilatation, resulting in a widening of the orifices between the orifices and ventricles. Aneurismal dilatations of the aorta or pulmonary artery just above the valves may lead to the same condition of incompetency in these valves. Where the valvular ring is too small it is probably due to cicitricial contraction following an earlier inflammation of the endocardium. This condition can usually be termined easily by simple examination.

## XCI.

### Lesions due to alterations in the valve segments.

The second great class of valvular lesions, as already mentioned, are caused by changes in the segments themselves

rendering them unable to close the openings to which they are attached. This condition of affairs is always the result of a previous inflammation of the endocardium, either of the segments themselves, or as in the case of the mitral and tricuspid, of the cordae which hold them. Where such an inflammation has existed the segments are permanently injured and subsequent contraction so distorts them that closure of the orifice is impossible. A segment may in like manner become adherent to the endocardium in its immediate neighborhood and so remain fixed, closure may be rendered impossible by the presence of vegetations or small masses of fibrin attached to the margins of the segments or calcarious deposits in the valves or in the vessels just above them may lead to the same result.

## XCII

### Simple Endocarditis.

When the condition of acute endocarditis is met with we find that the membrane lining the cavities of the heart is affected chiefly in the vicinity of one or more of the valves. The condition is characterized by some thickening and by the presence of small "vegetations" chiefly upon the valve segments, and also at times upon the ventricular lining. There is also some capillary injection.

## XCIII.

### Ulcerative Endocarditis

By far the greatest damage is done to the valve segments by that more severe form of endocardial inflammation, malignant, or ulcerative endocarditis. When this condition is present whole segments may be eaten away, or if not totally destroyed they may be perforated, or partially removed. In either case grave damage is done and the valve rendered incompetent.

## XCIV.

### The heart muscle.

This may be altered by a variety of morbid conditions. After attacks of fever of some duration, or in conditions of septic intoxication the heart muscle assumes a somewhat lighter color, is opaque and appears to be swollen; the condition being spoken of as "cloudy swelling." In cases of general anaemia the heart muscle shares with the rest of the body the anaemic condition and is decidedly paler than normal. When the anaemia is of long duration very severe, or when that disease known as pernicious anaemia is present, not only is the myocardium pale, but it assumes a more yellowish tint, is decidedly flabby, and is softer than normal. Here fatty degeneration has taken place, a portion of the muscle fiber being converted into fat. (See Lecture Notes on Fatty Degeneration.)

The fatty change may not be present everywhere, but may affect areas of heart muscle. Beyond doubt, the presence of these beneath the endocardium gives to the inner aspect of the heart a mottled appearance.

## XCV.

### Fatty Infiltration.

Quite different from the fatty change just described is that found in cases of general obesity. Here there is an accumulation of fat about the heart beneath the visceral pericardium, in the auriculo-ventricular groove and about the coronary arteries. Microscopic examination will disclose the presence of fat droplets within the muscle fibers at times, but the fat so present is not the product of the destruction of the cell protoplasm, it is simply deposited within the cell and under favorable conditions will be removed, while the fat present in cases of fatty degeneration, if removed, leaves a permanent

injury to the cell. In the condition of fatty infiltration the muscle has not lost its tone and is not much altered in color

## XCVI.

### Brown, or Pigmentary Atrophy.

We sometimes meet with hearts that are comparatively small, the walls of which are thinner than normal and the muscle darker in color, of a peculiar dusky brown hue. This condition is known as brown or pigmentary atrophy and is most frequently associated with such wasting diseases as cancer, etc.

## XCVII.

### Fibroid Induration.

In individuals past middle life, and especially in those who have thickened arteries and general fibroid changes in various parts we not infrequently find the heart muscle much firmer and not so deep red in color as usual. In many of these cases there has been a previous attack of pericarditis and upon examination bands of fibrous tissue can be seen running into the heart muscle between the bundles of muscle fibres. This may occur quite generally or be only found in a few places. As the process extends these bands of fibrous tissue encroach upon the muscle fibres to such an extent that they become atrophied and their place occupied by connective tissue. This change constitutes the lesion in Fibroid Disease of the heart.

## XCVIII.

### Hypertrophy.

Large hearts are very frequently met with and are due to a variety of causes that cannot be discussed here for lack of space. It may be stated however that they are found associated chiefly with valvular disease of some standing, renal disease, pulmonary emphysema and congenital stenosis of the pulmonary artery and aorta.

## XCIX.

*Varieties of Hypertrophy.*

Cardiac hypertrophy may appear in three different forms, the relative frequency of occurrence as in the order given.

(1.) Hypertrophy associated with dilatation of the ventricular cavities, or "Eccentric Hypertrophy."

(2.) Hypertrophy without dilatation, or "Concentric Hypertrophy."

(3.) Hypertrophy associated with a decrease in the size of the ventricular cavities, or "Cornified Hypertrophy."

According to some authorities the last mentioned form of hypertrophy never occurs, the apparent, as the size of the ventricular cavities being due to the contraction produced by rigid rigor mortis.

For the weight and measurements of the normal heart see the Table of weights and measurements in the last chapter.

Of the other changes met with in the heart muscle may be mentioned:-

(1.) Small abscesses.

(2.) Tubercles.

(3.) Ulcerations and small anurisms

(4.) Syphilitic gummata.

## C.

*The coronary arteries.*

The general condition of the coronary arteries should be noted, especially with reference to the presence of degenerative changes of the vessel walls. It is not at all uncommon to find in elderly people extreme atheroma of these vessels which then appear as tortuous, rigid elevations on the surface of the ventricles. The openings of the coronary veins in the right auricle just above the auriculo-ventricular orifice should be examined and its size noted. In like manner examine the openings of the coronary arteries in the aorta just above the segments of the aortic valve.

## CI

### Embolism of the Coronary Arteries.

Where death has occurred suddenly and without apparent cause the coronary arteries should be carefully examined to as great extent as possible. Open them with a pair of small scissors and be on the lookout for the presence of an embolus. Sudden closure of one of the larger coronary arteries by an embolus results in sudden death.

## CII.

### The aorta.

Not only should the aorta just above the aortic valve be examined, but both the thoracic and abdominal portions should be opened and their condition noted.

One frequently finds, just above the valve a larger, or smaller number of yellowish patches in some of which may be found calcareous particles. These patches are areas of fatty degeneration of the intima of the vessel and in this degenerated tissue lime salts are sooner or later deposited, leading to the formation of plates which are often of considerable size and quite numerous. One of the possible results of this condition is rupture of the aorta and sudden death. The formation of these calcareous plates is not confined to the beginning of the aorta, but may extend throughout its entire course, even involving the greater portion of the arterial system. One should therefore while examining the thoracic and abdominal aorta open the chief arterial trunks which it gives off and examine them also.

## CIII.

### Aneurism.

It occasionally happens that through some weakness of the coats of the aorta it begins to give way gradually to the pressure of the blood within it, and slowly dilates.

Since the weakness is usually confined to a limited area this dilatation is confined to the weak spot and gradually forms a pouch in the wall of the aorta, often of considerable size. This pouch is spoken of as an aneurism and is most frequently found in the ascending portion of the vessel a little above the valve, and usually outside of the pericardium. It is also found in a large number of cases in the arch of the aorta. When such an aneurism is found it should be carefully opened and the condition of the walls noted. Usually, because of the more rapid circulation within the expanded portion, and because of the changes in the vessel walls, the blood will be found to have coagulated along the sides, and a careful inspection will show that the clot is arranged in layers. It is by this process of clotting that the aneurism is prevented from rupturing for some time. Sooner or later however the walls give way and death rapidly ensues. Where an aneurism is found the condition of the parts against which it has pressed must be carefully examined. It will usually be seen that where the ribs or the sternum have been pressed upon the bone will be to a considerable extent absorbed. In rare cases this absorption is carried to such an extent that when the aneurism ruptures it breaks externally through the chest wall, death resulting rapidly from hemorrhage.

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# Chapter X.

## The Liver, Gall-Bladder and Bile Ducts. The Spleen, The Pancreas.

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## The Liver.

**CIV.**

*The Normal Organ.*

Under normal conditions the weight of the liver is about

50 - 60 ounces, or roughly speaking from three to three and a half pounds, in the adult.

For a description of the conditions under which the weight of the liver is increased or diminished, see the section on diseases of the liver in the body of the manual.

In infants the liver is relatively larger than in adults, and the size of the two lobes is more nearly equal. As in the case of the spleen and some other organs, one occasionally meets with small accessory pieces of liver tissue, or with small extra lobes. These are of no import.

The liver is covered with a close fitting capsule, composed of two layers of tissue, the outer of which is simply a reflection of the peritoneum, while the inner is the true capsule, or the capsule of Glisson as it is sometimes called. This true capsule sends numerous prolongations into the liver substance, along with branches of the hepatic artery and portal vein. These structures together with branches of the bile ducts constitute what are known as the portal canals of the liver.

## CV.

*Roughening of the surface.*

Normally the capsule is smooth and glistening, but it may become roughened in any of the following conditions. (1.) The various forms of cirrhosis. (2.) Fatty degeneration, and acute yellow atrophy. (3.) Malignant disease. (4.) The late stages of extreme passive congestion, when on account of the shrinkage of the organ the capsule becomes wrinkled. (5.) Any condition that leads to a considerable decrease in the size of the organ. The surface may also become roughened because of previous inflammation of the peritoneal covering. This may occur generally over the surface of the organ, or may be found only in patches here and there. In consequence of such inflammation the organ

may be found to be adherent to surrounding parts, especially to the diaphragm, the hepatic flexure of the colon, to the duodenum, or to the head of the pancreas. Where the capsule of the liver has been thus inflamed and thickened a condition known as "Perihepatitis" is produced.

## CVI.

*Mottling of the surface*

In a large percentage of autopsies the surface, and especially the anterior surface, of the liver is seen to be mottled, either by whitish areas, or by patches of a dark reddish purple color. In the first instance the spots are due to anaemia from pressure through the abdominal walls, exerted after death in some way. The darker spots are due to chemical changes produced by the action of sulphuretted hydrogen upon the small quantity of urine contained in the liver. These spots are found where the distended stomach, or colon has been in contact with this organ.

The under surface of the liver may be of a very deep reddish-purple color, on account of hypostatic congestion.

## CVII.

*The Gall-Bladder.*

The liver should now be turned over and the gall-bladder examined. Usually this is more or less distended with bile. If any adhesions are present they should be noted. With a pair of scissors open the gall-bladder, beginning at the fundus. Allow the bile to escape, and note its quantity, consistency and color. It is usually thick and of a golden brown tint. Concretions may not infrequently be present and should be counted and saved for future chemical examination. Examine the opening of the cystic duct to be sure that no small calculus has worked its way into it.

Where malignant disease of the gall bladder is found, such as primary cancer, etc., the organ is found to be greatly

increased in size, to be firm in consistency and of small calibre. The association of this condition with the presence of gall stones should be sought for and the bile ducts traced as far as possible in order to determine how far the new growth has extended.

**CVIII.**

For the changes of the liver in the following conditions refer to the section on Diseases of the Liver in the body of the Manual.

(1.) Cloudy Swelling
(2.) Chronic Passive Congestion
(3.) Fatty Infiltration
(4.) " Degeneration
(5.) Amyloid Infiltration
(6.) Atrophic Cirrhosis
(7.) Hypertrophic Cirrhosis
(8.) Syphilis " Congenital.
(9.) " Gummata
(10.) Tuberculosis
(11.) Typhoid Fever
(12.) Abscess.

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# The Spleen.

**CIX.**

*Size Weight, etc.*

The normal weight of the spleen is from six to seven ounces. In elderly people however it is considerably less on account of the atrophy that normally takes place as age advances. When found to be smaller than normal its capsule is wrinkled and has lost its usual shiny appearance. Occasionally small bodies composed of splenic tissue are found either attached to some portion of the organ, or independent, in the gastro-splenic omentum. These are known as "accessory spleens."

**CX**

*Lamellar Fibromata.*

While the capsule of the spleen is normally smooth and has the same glistening appearance that characterizes the coverings of all the abdominal and thoracic organs, one

fonds at times that it is more or less roughened by firm nodular masses, hard to the feel and almost cartilaginous in consistency. These masses are composed of dense fibrous tissue and are strictly confined to the capsule. To them the name of Lamellar Fibromata has been given.

**CXI.**

*Infarct Scars.*

The usual smooth surface of the spleen may also be found to be indented at times with scar-like depressions. Upon section of such an area an abundance of connective tissue is found, the contraction of which has led to the irregularity of the surface. These masses of connective tissue represent the seats of old splenic infarcts. In recent infarcts there are no depressions but areas of deep congestion, the peritoneum over which may be inflamed.

**CXII.**

*Discoloration.*

As in the case of the liver the surface of the spleen may be discolored by patches of a deep purple-green hue, which in some instances are so dark as to be nearly black. Careful examination in these cases will show that the spleen has been in contact with the stomach, and that the sulphuretted hydrogen gas so often contained therein has acted upon the iron in the splenic tissue forming in the tissue in contact with the stomach the sulphide of iron which has discolored the surface of the organ.

On section the spleen is normally of a deep red color which turns to a bright red as the oxygen of the air acts upon the haemoglobin of the blood in the exposed surfaces.

**CXIII.**

A description of the spleen in the following conditions will be found in the section devoted to the Diseases of the Spleen in the body of the Manual.

(1.) Complete Infiltration (3.) Tuberculosis
(2.) Chronic Passive Congestion (4.) Typhoid Fever.
(5.) Leucocythemia.

# The Pancreas.

## CXIV.

### The Normal Organ.

The normal weight of the pancreas in the adult is from three to four ounces. In consistency it is usually only moderately firm, and is very loose in structure. As a rule the pancreatic artery should be opened with a pair of small scissors and its condition noted. The pancreatic duct should be examined in the same way, as occasionally one finds calculi occluding one or more of its branches. To reach the pancreatic duct carefully remove the substance of the organ at about the junction of the head with the body of the same till a white tube is reached. This is the duct and it may now be followed forward and backward and its main branches investigated.

## CXV.

### Fat Masses. Malignant Growths.

In the interstitial tissue of this organ, which is abundant and easily observed, little opaque spots are sometimes seen. These are small collections of fat and are of no importance.

Malignant growths when occurring in the pancreas are most frequently located in the end nearest to the liver, that end known as the head of the organ. When such growths are found notice whether the mass presses on the common bile duct, for when there is jaundice it is sometimes explained in this way. Primary malignant growths in the pancreas sometimes give rise to metastatic growths in the liver, and where neoplasms are found in the latter organ the former should be carefully examined for the same condition.

CXVI.

*Haemorrhagic Pancreatitis*

The pancreas is very rarely found to be greatly softened and to be infiltrated with blood. The condition is one of acute haemorrhagic inflammation and is known as haemorrhagic pancreatitis.

CXVII.

*Abscesses.*

Occasionally small abscesses of the pancreas are found, which in a certain percentage of cases are associated with impaction of the bile ducts with gall stones.

CXVIII.

*Cirrhosis.*

For a description of this condition see the section on the Pancreas in the body of Manual

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# Chapter XI.

## The Kidneys and Ureters

CXIX.

*Weight, etc.*

In the normal adult the weight of the kidney is about six ounces. The left kidney usually weighs somewhat more than the right. Where there is but one functionally active kidney it becomes increased in size and weight, and may equal the combined weight of the two normal organs.

CXX.

*Opening the Kidney.*

In order to make a careful examination of the kidney it must be opened by an incision along its outer margin carried entirely through it to the pelvis. This portion of the organ in the healthy adult is smooth and shiny

and shows but few injected vessels. Into it the pyramids of the organ dip down and open by minute orifices. From it the ureter leads away to empty into the bladder.

## CXXI.

### Removal of the Capsule.

After having examined the condition of the pelvis the capsule of the kidney should be removed. This is done by catching it between the thumb and finger along the edges of the incision just made. Normally it is stripped away from the organ without any difficulty, disclosing the underlying kidney substance. Occasionally in certain morbid conditions the capsule is adherent to the organ and in attempting to remove it small portions of the kidney substance are torn away with it. This is seen especially in the kidney of interstitial nephritis, where small bands of connective tissue lead from the capsule into the organ and mingle with the overgrowth of this tissue which forms the chief lesion in this disease.

In adults the normal kidney surface is smooth. In children, and not infrequently in adults some remnants of foetal lobulation are visible.

## CXXII.

### Color of the Kidney.

Normally the cut surface of the kidney is of a dark red color, the medullary substance presenting a striated appearance on account of the long straight tubules. The organ may present on its surface discolored spots as mentioned in speaking of the spleen and liver, and due to the same cause. A number of conditions alter the color of the organ. It is much lighter in such diseases as anaemia, fatty changes, amyloid infiltration, and chronic interstitial nephritis, while the vessels containing blood stand out more prominently against the lighter back ground. The pyramids, as a usual thing are more deeply congested

than the cortex, and therefore appear sharply defined from it. The kidney is of a darker color in such conditions as acute nephritis and in congestion.

## CXXIII.

*Inflammation of the Pelvis*

When the pelvis of the kidney becomes inflamed it loses its smooth and glistening appearance, becomes roughened and opaque, particles of fibrin may be found attached to its walls and its vessels become injected. In very severe cases ulceration may take place, or its cavity may become partly filled with pus. In these cases pus will be found in the urine.

## CXXIV.

*Infarcts, Old and Recent*

The surface of the kidney which is normally smooth may, at times present scar-like depressions, to the bases of which the capsule will be found to be adherent. These depressions are due to previous infarction. Between the depressions the surface of the kidney is often considerably elevated, and where the scars are numerous it may give to the organ a roughly granular appearance, not unlike that found in the condition known as "granular kidney."

While in most other organs infarcts are haemorrhagic in character on account of their open structure, (See Infarction, Lecture Notes.) in the kidney they are anaemic, or white, and are seen to be surrounded, when studied in section, by a zone of engorgement, and small cell infiltration.

## CXXV.

*Pyemic Infarcts.*

Aside from the simple anaemic infarcts just mentioned, which are due to the usual causes of infarction, (See Lecture Notes), one occasionally meets with what are termed "pyemic infarcts." These are small, and often appear as congested spots with their bases, as in other infarcts, toward the cortex

of the organ, in which suppuration is beginning about their centres. When this suppurative process has advanced these infarcted areas are converted into multiple abscesses.

## CXXVI.

### Cysts.

Another condition of the kidney more frequently met with than infarction is cyst formation. In many cases a few small cysts are found on the surface of the organ which is otherwise comparatively normal. When these are examined they are found ~~they are found~~ to contain a clear, yellowish fluid, and to have smooth walls. They are probably due to some occlusion of a uriniferous tubule, the excreted urine being retained, and gradually forming for itself a small cavity, by continuous pressure on surrounding parts, which upon closer examination are found to be atrophied. From this mode of formation these structures have been called "retention cysts".

Not infrequently in cases of chronic interstitial nephritis numerous cysts are formed, at times so converting the organ into a mass of these structures that the condition is spoken of as cystic disease of the kidney. In these cases the contents are not always watery, but may be somewhat thick, on account of the presence of degenerating epithelial elements. Where the watery portion of the contents becomes absorbed as is sometimes the case the contents becomes cheesy in consistency, and one might be led to think that he was examining an area of tissue necrosis which had undergone cheesy degeneration. The formation of these cysts in cystic disease is practically the same as that of the retention cysts, already mentioned, bands of connective tissue by contracting and thereby closing up the tubules, prevent the further on flow of the urine which in consequence accumulates above the constriction.

## CXXVII.

*Hydro-Nephrosis.*

In the condition known as hydro-nephrosis cyst like cavities are found which upon close examination are seen to be but dilated portions of the calyxes of the pelvis and open into it

## CXXVIII.

*The Cortex.*

In estimating the thickness of the cortical substance of the kidney it must be remembered that under normal conditions it may vary somewhat within narrow limits, and that if the section be made obliquely it will appear to be broader than it really is.

The cortex is found to be thickened in the following conditions. (1) Fevers, especially of the septic variety. (2) Acute parenchymatous nephritis, and (3.) Chronic parenchymatous nephritis.

In the following conditions it is narrower than normal;- (1) Hydro-nephrosis. (2.) Chronic interstitial nephritis. In using the term chronic interstitial nephritis we here include the contracted white kidney, and the small red kidney of arterio sclerosis.

## CXXIX.

*Other Morbid conditions of the Kidney.*

For the gross appearances in the following morbid conditions consult the section on diseases of the kidneys in the body of the manual.


(1.) Chronic Passive Congestion page 92
(2.) Acute Parenchymatous nephritis " 95
(3.) Chronic " " " 96 to 98
(4.) " Interstitial " " 99 " 102
(5.) Amyloid disease of the kidney " 103 " 105
(6.) Tuberculosis of the Kidney " 108 " 109

## The Ureters.

### CXXX.

The ureters should be dissected away from surrounding structures and traced from the pelvis of their respective kidneys to the bladder which they enter in an obliquer manner, one on each side. These tubes lie, one on each side, close to the psoas muscle, and can be found without difficulty. Any of the following conditions may be present and should be looked for.

(1.) Twists
(2.) Dilatation, general or local
(3.) Strictures
(4.) Inflammation.
(5.) New growths.
(6.) Impacted calculi.
(7.) Ulcerations.
(8.) Thickening of the walls, etc.

# Chapter XII

## The Pelvic Organs.

### The Male and Female Organs of Generation

### CXXXI.

#### The Bladder.

This organ, the method for the removal of which has already been given, should be examined for any of the following conditions, which are so easily made out that a detailed description of them is not necessary.

### CXXXII.

#### New growths.

These may be found either growing from the outer side of the bladder wall, from the inner side of the wall, in either case forming a distinct mass, or infiltrating the walls generally, in which case no distinct tumor will be found, but the walls in the locality of the neoplasm will be greatly thickened. The margins of

the new growth will not be sharply defined, but it will shade off into the healthy tissue on all sides. New growths of the bladder are, in the majority of cases malignant in character, and carcinomatous in structure.

## CXXXIII.

*Dilatation.*

This condition is most frequently found in men in advanced life and is secondary to some trouble in the prostate gland, most frequently hypertrophy, or to some trouble in the urethra, generally stricture following attacks of gonorrhea in early life. Both of these primary conditions lead to a very gradual decrease in the size of the urethra and to the consequent very gradual damming back of urine in the bladder, the walls of which slowly lose their tone till finally there is considerable dilitation and the retention, constantly, of a certain amount of urine which is not expelled owing to the weak contraction of the bladder walls during the act of micturition.

## CXXXIV.

*Contraction.*

This is much less common than dilitation and is usually due to thickening of the bladder walls from one cause or another. Where there is a neoplastic infiltration of the walls, for example, the cavity of the organ will be much reduced in size, and though there is no actual contraction of the muscles, the cavity is contracted in size, and this is what is most frequently meant when contraction of the bladder is spoken of.

## CXXXV.

*Hypertrophy of the Walls.*

When this condition is present the muscle fibers show up quite distinctly as columns beneath the mucous

membrane, which is not infrequently so thrown into folds that small pouches are formed, in which calculi may become encysted. When hypertrophy is found carefully investigate the openings of the ureters, which may be very much obscured. Pass a probe from the cut ends of these tubes into the bladder, for occasionally small calculi will be found in that portion of the tube which passes through the muscular walls. When the bladder is found empty and contracted the mucous membrane will be thrown into folds, and the underlying muscle fibers so distinct at times that the condition may be mistaken for hypertrophy.

## CXXXVI.

### Inflammation of the Mucous Membrane.

In acute inflammation of the bladder, that is in acute cystitis, we find the mucous membrane deeply injected and covered with a fibrinous exudate. In very severe cases ulcerations are found here and there.

Where the inflammation is chronic in character the mucous membrane is thickened and thrown into folds. It is of a dirty greyish-purple color, is more or less covered with thick, tenacious mucous and at times contains a deposit of phosphates. Slate colored patches which are sometimes found, indicate areas previously inflamed

## CXXXVII.

### Tubercular Cystitis.

Tubercular disease of the bladder is characterized by the presence of ulcers of varying size, which are due to the breaking down of the tubercles that have formed in the mucous membrane. As the disease advances these ulcers increase in size, their bases are rough and their edges irregular. A microscopic examination discloses numerous tubercles in all

stages, in the walls of the ulcers and in the surrounding tissue.

## CXXXVIII.

*The Urine, &c.*

In examining the bladder always note the character of the urine that it contains. It is often turbid, but unless ammoniacal its turbidity is of no importance. It should be carefully examined for any trace of blood, pus, mucous or deposits of gravel. Urine that has stood in the bladder for some time after death does not of necessity contains albumen.

## CXXXIX.

*The Prostate Gland.*

Before concluding the examination of the bladder examine the prostate gland to see whether it is enlarged, and if so in what way, and whether it presses upon the urethra in such a way as to partly occlude it. Notice also whether any part of the base of the bladder is below the level of the urethra.

## CXL.

*The Urethra*

To examine the male urethra open the penis along its dorsal aspect with a pair of scissors. The mucous membrane should be examined for ulceration, cicatrization and for any signs of inflammation. The location of any stricture should be noted, and false passages looked for. Measure the circumference of the urethra above and below the stricture, and take also the length and circumference of the stricture itself.

## CXLI.

*The Testes.*

Leaving out of consideration the examination of the epididymis, cord, seminal vesicles and vas deferens,

which need only be examined in special cases and a consideration of which will be omitted from this manual for lack of space, the testis should be sectioned as was the kidney and its condition noted. In health its tissue is white, or creamy in color, bulges out after the section is made and has a fluffy appearance.

## CXL II

### Syphilitic Orchitis.

The most common morbid conditions found are due to syphilis. When this disease affects the testis we may have either a general development of connective tissue, rendering the organ firm and hard, or gummata may be formed. These latter structures appear as rounded areas of cheesy material, sharply defined from the healthy tissue, and are surrounded by a more or less diffusely developed zone of connective tissue.

## CXL III.

### Tubercular orchitis.

In cases where the testicle is affected with tuberculosis, cheesy masses may also be found not only in the testicle, but also in epididymis, seminal vesicles and cord. These resemble very closely the gummatous masses just spoken of, but are usually not so large, and when found in the testicle the epididymis and cord will show numerous small tubercles, which are easily recognized. It may however have a microscopic examination to determine the nature of the cheesy masses found in the substance of the testis.

## CXL IV

### New Growths.

Tumors of the testis are most usually malignant in character. Sarcomata are most frequently met with. When starting in the testis they spread up the cord

and involve the lumbar glands. A non-malignant tumor composed of cartilage (enchondroma) is also at times met with in this organ.

## CXLV.

### The Female Organs of Generation.

This group of organs, omitting those that are external, consists of the vagina, uterus, tubes and ovaries.

## CXLVI.

### The Vagina.

It is not necessary to measure this organ. The following points are to be observed:-

(1.) Are there any lacerations

(2) Are there any ulcerations of the mucous membrane.

(3.) Is there any discharge and if so what is its character, and is it from the vagina or from the uterine os.

(4.) If deemed necessary the vaginal discharge should be examined for gonococci, or spermatazoa.

## CXLVII.

### The Uterus.

The uterine cervix extends into the upper portion of the vagina. The condition of this cervical portion and of its os varies according to whether the woman has, or has not borne children. In the latter case the os is round and shows no signs of laceration, its edges are smooth. In those who have borne children the os is irregular in shape, is usually slightly scarred where portions have given away during labor, or may show either considerable laceration, or the scars from the same.

## CXLVIII.

*Granular Erosions.*

Where there is considerable catarrh of the cervix the mucous membrane may be roughened and granular in appearance. This condition is spoken of as erosion of the cervix

## CXLIX.

*Malignant Growths.*

When malignant growths of the uterus occur they most frequently start in the cervix, and may be found upon examination as uneven masses, not infrequently showing some ulceration of their surfaces, and sometimes presenting a cauliflower appearance. Mucous polypi may exist in the cervical and extend into the vagina through the external os.

## CL.

*Opening the Uterus.*

In order to open the uterus for further examination incise the organ along its anterior aspect from the fundus to the external os. Extend the incision from its upper end into the Fallopian tubes on either side.

## CLI.

*Examination of the uterus.*

Normally the uterus is small (2½ in. in length) firm and contains a small triangular cavity lined with smooth walls. In advanced life it undergoes atrophy. During menstruation it, as well as the vagina, contains a little dark blood, and the uterine mucosa is shreddy. The following conditions should be looked for, and if found, noted:

## CLII.

(1) *Pregnancy.*

**CLIII.**

(2) Endometritis.

Here the mucous membrane is usually roughened, thickened, nodular, and may be covered with a layer of pus. In other cases it may be the seat of numerous small polypi.

**CLIV.**

(3). Fibroid Tumors.

These may be found either upon its outer aspect beneath the serous coat (sub-serous), within the muscular wall of the organ, (intra,-mural), or beneath the mucous membrane, (sub-mucous). The sub-serous and sub-mucous tumors may be either sessile or pedunculated. Malignant tumors rarely start from the body of the organ, but from the cervical portion in the vagina.

**CLV.**

(4) The changes produced by septic (puiperal fever.

In cases of puiperal fever the uterus is always larger than normal as involution has not had time to take place. Its walls are thickened and swollen and its cavity contains a mass of necrotic material, the remains of placental tissue and of the sloughing diseased uterine mucosa. There is not infrequently a general peritonitis and the pelvic cavity may contain considerable pus. The condition of the uterine mucous membrane, which in some cases is not as necrotic as just described, but is smoother and covered with a bloody discharge, is due to the action of different forms of micro-organisms, chiefly the streptococcus and less frequently the Bacillus Coli Communis. Cultures should be made from the necrotic material.

**CLVI.**

The Fallopian Tubes.

Examine the fimbriated extremities of the Fallopian tubes

and determine whether they are patent, or have been closed by some previous inflammatory process. Note also whether the tube is free along its entire length, or is adherent to surrounding parts. See whether any changes in the calibre of the tube have occurred and look especially for localized enlargements which may contain either a watery fluid, (Hydrosalpynx); pus, (Pyosalpyn), or blood (Hematosalpyn). Where these conditions are present there are signs of old, or recent inflammation in the surrounding parts. Tumors of the Fallopian tubes are rare.

## CLVII.

### Haematomata.

Between the folds of the broad ligaments occasionally one finds a large collection of blood, firmly clotted and forming a tumor mass frequently of considerable size. The collection of blood may occur as a single mass, or there may be a number of similar collections, it may be confined to one side or occur on both. These collections of blood between the folds of the broad ligaments are known as haematomata. At times they are encapsulated.

## CLVIII.

### The Ovary.

In the normal adult female the ovary is a flattened, almond shaped organ, lying, one on each side, near the end of the Fallopian tube in the broad ligament. After maturity the surface of these organs is scarred, each scar representing the healed corpus luteum from which at some previous menstrual period an ovule has been discharged. Before the age of puberty the surface of the ovary is smooth. In advanced age the organ atrophies and becomes very firm and hard.

In examining the ovary its position should be first determined, as it is not infrequently found to be prolapsed and lying in Douglas pouch.

Enlargement of the ovary may chiefly be due to new growths, many of which are malignant in character to inflammatory processes, to tuberculosis and to the formation of cysts. The presence of any of these conditions should be noted.

## CLIX.

### Section of the ovary.

Upon section the ovary is seen to be bounded by a narrow layer of dense connective tissue, the "Tunica albuginia". The central portion of the organ is composed of a looser stroma containing many blood vessels and beneath the outer tunic can be seen numbers of Graafian follicles in all stages of development, the largest being nearest the surface and often containing a clear fluid. Corpora Lutea, the bodies that remain after the Graafian follicles have discharged their contents can also be seen as small areas of a dark brownish color. The corpus luteum of pregnancy is always much larger than that of menstruation. The oldest Corpora lutea are merely small collections of pigment.

## CLX.

### Inflammation of the Ovary.

In inflammation of the ovary the organ is found to be increased in size, is of a reddish color and is softer or consistency than normal. In pus it may become as large as a pigeons egg or even larger and upon section may contain small collections of pus.

## CLXI.

### Cysts.

One must be careful in examining the ovary, not to mistake the large Graafian follicles, and the cysts formed from old corpora lutea for true ovarian cystic degeneration. Cystic disease of this organ is not [illegible] and is found

inflammation (chronic ovaritis) cystic degeneration often occurs, numerous, small, fluid containing cavities being found scattered throughout the organ.

Space will not permit us to give here a description of the various forms of cysts and of cystic tumors that are found in the ovary, and the student is therefore referred to larger works on the subject.

## CLXII.

### The Female Breasts.

In making the external examination of the female breasts the following points must be observed :-

(1.) Size. Remember that this varies both with age and during gestation and lactation. Hypertrophy occurring at other times is due either to glandular increase, or to an increase in the amount of adipose tissue.

(2.) Consistency

(3.) Color of areolae.

(4) Condition of the nipples.

(5) Presence of nodular masses.

The breast is now to be sectioned, and this should always be done from the upper side, being careful not to injure the skin. The following conditions should be sought for.

## CLXIII.

### Acute Mastitis.

Acute inflammation of the breasts is characterized by the following appearances. The tissue is congested, swollen, more or less infiltrated and there are not infrequently abscesses formed. The situation of these abscesses varies in different cases. They may be found either in the substance of the gland, behind the gland, that is between it and the pectoral muscle, or just beneath the skin.

## CLXIV.

### Chronic Mastitis

Chronic mastitis is characterized by an overgrowth of connective tissue in the gland. The condition may be either general or local and is not infrequently accompanied by the formation of cysts. Occasionally giant fibrous nodules are met with. The cysts above mentioned, which may also be found at times in the atrophied breasts of old age, may contain either altered milk, serous fluid or more rarely blood.

## CLXV.

### Tumors.

Tumor masses are frequently found in the female breasts and either benign or malignant. Aside from the fibrous nodules just mentioned as occurring in chronic mastitis, adeno-fibromata, sarcomata, adeno-sarcomata and carcinomata are met with.

Whenever a tumor is found in the breast no matter what its location, or its apparent character a careful examination of the axillary glands should be made to discover whether or not extension to these structures has taken place.

In describing a tumor of the breast note (1) whether or not it is encapsulated (2) What its consistency is. (3) Whether it is attached to the skin or is freely movable (4) Whether or not the nipple has become retracted, and (5) Examine also the underlying pectoral muscle for possible metastatic growths.

## CLXVI.

### Color of the Breasts.

Normally where the gland is in a resting state the color of the cut section is whitish on account of the preponderance of fibrous tissue. Close observation shows, here and there, small pinkish spots, the sections of glandular acini.

During gestation and lactation the color of the sections is more pinkish and looks granular. On pressure at this

time a small amount of colostrum, or of milk can be squeezed out, which, unless examined carefully, may be mistaken for pus.

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# Chapter XIII.

## The Skull, Dura and Cerebral Sinusses

### CLXVII.

#### The Skull Cap

The examination of the skull resolves itself into an examination of the skull cap that has already been removed, and an examination of the skull.

The chief things to be looked for after carefully removing the dura are, (1) Fractures. (2) Peculiarities of development. (3) Morbid changes in the bone, and (4) Tumors. In children the fontanelles should be noted. The anterior fontanelle should be closed by the end of the twelfth month, the others usually open for a somewhat longer time. Should these openings continue patent for a longer time than is normal it usually indicates either rachitis, or hydrocephalus.

### CLXVIII.

#### Changes in the Bone, Thinning and Softening.

Thinning of the bones of the skull is sometimes found and may have advanced to such an extent that only a delicate bony structure is met with instead of the usual firm thick bone. This destruction of bone is most frequently due to congenital syphilis and to rachitis. In the adult, heavy tumors of the scalp will in some cases lead to an absorption of the bones of the skull, on account of the constant pressure that they exert. Hydrocephalus also produces thinning of the bones of the skull because of expansion.

Softening of the bones of the skull sometimes occurs as a

result of syphilis. Also these cases the bones may be easily cut with a knife.

## CLXIX

### The Periosteum. Chronic Periostitis

The condition of the periosteum of the cranial bones must also be carefully examined. General thickening of these bones is the result of chronic periostitis, and is not infrequently syphilitic in origin, either congenital or acquired. The changes produced may be tabulated as follows:-

(A.) Congenital Syphilis

(1.) Atrophic changes. (Craniotabes), with marked thinning of the bones, or

(2.) Softening without thinning of the bones, or

(3.) Changes leading to the formation of symmetrical nodes (parostitis) over both parietal and frontal bones, around the anterior fontanelle

(B.) Acquired Syphilis

(1.) Local thickening due to periostitis. (nodes)

(2.) General sclerosis of the calvaria due to ostitis

(3.) Carious and ulcerative changes. The bones become worm eaten and pitted, and at the same time new bone is formed by the periosteum, giving the surface a reticulated appearance. Simultaneously, there is sclerosis and areas of dead bone may be seen

## CLXX.

### The Base of the Skull.

Fractures should be looked for. Neuroses and inflammatory lesions especially about the petrous portions of the temporal bone may occur. In these cases the dura is dull and lustreless

## CLXXI.

### The Cerebral Sinuses.

(1.) Open the cranium, fixing and [illegible] of the [illegible]

(2) Trace the petrosal sinusses backward, the larger one, the inferior to the jugular fossa, and the smaller one, to the lateral sinus.

(3) Open the lateral sinus and examine it for thrombosis, bearing in mind the difference between the clots that are formed before and after death. When clots are found that have existed before death they are due either to meningitis, to an inflammatory process of the internal, or middle ear, or to injury to the bone.

(4) Where thrombi are found the jugular veins, cerebral veins and the superior petrosal sinus should be carefully examined for similar changes.

(5) Where thrombi are found one should also look for such later changes as softening, and the condition of the walls of the vessels should be noted.

## CLXXII.

### The Pituitary Body.

The Pituitary body left in the Sella Turcica, should be examined, especially if Acromegally, or pathological changes in the thyreoid have existed, as it is now supposed that in these conditions this structure is altered.

## CLXXIII.

### The Dura Mater.

Examine the dura mater for the following points:

(1.) Adherent dura. This frequently due to old inflammations occurring as a result of syphilis, or alcoholism. Other evidences of these conditions should be looked for in other portions of the body.

(2.) Blood effusion. When this condition is found between the dura and the skull it is an induction of death from violence and the bones of the skull should be very carefully examined for fracture, and the scalp for evidence of a wound of any kind.

(3.) Purulent effusion. The presence of pus between the dura and the skull is of rare occurrence and in the majority of cases is due to some disease of the bone which should be looked for.

(4.) Thickening of the dura. This is rarely found and is due to the development of a membraneous tissue, (Pachymeningitis, due to syphilis, alcoholism, and found in the paralyzed insane.

(5.) Pachionian Bodies. On either side of the longitudinal fissure there are not infrequently found numerous minute rounded white bodies that resemble tubercles, but must not be mistaken for them. These are known as Pachionian bodies.

**CLXXIV.**

Before completing the examination of the membranes of the brain, carefully inspect the Arachnoid membrane and the pia mater for evidences of meningitis.

The veins running over the surface of the brain are frequently noted as being full of blood. This is not pathological and does not indicate ante-mortem passive congestion.

## Chapter XIII. (a)
## Examination of the Brain.

**CLXXV.**

The surface of the Brain.

The surface of the brain should be examined for the following conditions; (1.) Atrophy of the cortex. (2.) Asymetry. (3.) Local prominences. (4.) Depressions (5.) Blood clots outside of the arachnoid. (6.) Areas of softening (7.) Injuries, bruises, etc., which are most severe opposite to the external injury.

Local prominences when found may be due to the presence of tumors or of abscesses, while depressed areas are usually the result of pressure exerted from without and the skull should therefore be carefully examined for tumors, or areas of depressed bone.

Where areas of softening are found in the cortex the pia mater will often be found to be adherent over the same. On attempting to remove the membranes one finds that it is pealed off with much more difficulty than that over the unimpaired parts

Atrophy, suppurative abscess formation and the presence of a tumor must be carefully looked for in those cases that have presented symptoms indicating the involvement of cortical areas

In cases in which bruises of the surface of the brain are found, the skull must be examined for injury. Opposite to the point of injury to the surface of the brain, one will usually find either fracture, or severe injury to the skull, and beneath this point of injury the brain may also have sustained some damage.

## CLXXVI.

### Meningitis.

In every examination of the brain the operator should look carefully for the signs of meningitis. Generally the surface of the organ will be found to be bathed with a purulent, or sero-purulent fluid, which being external to the pia mater can be readily brushed off. The process may be either general or local. Such a condition as this is usually secondary to inflammations of other parts such as the nose, ears, etc, or it may be the result of direct injury or of tumor, or abscess formation. In looking for the cause of this inflammation of the meninges one should carefully examine the organs first mentioned if there is no sign of fracture or other condition that could have produced it.

## CLXXVII.

*Primary Meningitis, Spinal Cord - Spinal Meningitis*

A second variety of meningitis differing somewhat from that just described, may be found. In this variety the inflammatory exudate of pus, or sero-purulent fluid is generally more intimately associated with the pia mater, and in consequence is not so easily washed away. The inflammation in these cases is primary and not due to extension from other inflamed parts. As in every case of meningitis the arachnoid membrane becomes opaque and dull. In severe cases there will be found softening of the cerebral cortex and hemorrhages may be met with here and there in the brain substance.

## CLXXVIII.

*Tubercular Meningitis.*

A third variety of meningitis is that due to the tubercle bacillus. In this variety the signs of inflammation, such as were met with in the other varieties are wanting. There is no exudate of pus, or of sero-purulent fluid, but the pia mater is found to be studded with miliary tubercles. These as a usual thing are most numerous along the course of the vessels at the base of the brain, near the optic chiasm, and over the lower portions of the cerebral surfaces generally.

## CLXXIX.

*Chronic Meningitis*

A chronic inflammation of the meninges is occasionally met with, especially in old alcoholics, and in syphilitic patients. In the former the condition is apt to be general, while in the latter it is most frequently local. Chronic meningitis may occur in children and is found chiefly at the base of the brain. In all cases of this condition the pia mater is found to be opaque and thickened.

## CLXXX.

*The Cerebral Vessels. Atheroma, etc.*

The vessels of the brain should always be examined carefully for signs of atheroma and for calcareous changes. Where atheroma is found the vessels are rigid, do not collapse when cut across, and can be felt to be distinctly thickened.

When carefully examined it is seen that the intima is considerably thickened, and in places separates easily from the other coats of the vessels. So great may this thickening be that the lumen of the vessel may be nearly entirely occluded. This condition of atheroma is most frequently, though not always, a result of syphilis. Aside from examining the vessels for this condition, one should look also for thrombi, emboli or aneurismal dilitations.

## CLXXXI.

*Section and examination of the brain.*

So far we have only examined the surface conditions that may be found. In order to continue our examination we must before proceeding further, section the organ. Unless the brain is sectioned in an orderly and intelligent manner it is useless to continue the examination, as the normal relations of the parts, which are complicated in the extreme, are so altered that one is unable to localize any morbid conditions that may be found. Two methods for examining the brain are chiefly used, both having their especial advantages, the one to be used under certain conditions, the other to be used when different conditions present themselves.

One of these methods was introduced by the German pathologist Virchow and is known as Virchow's method, the other being first advocated by Petres, and afterward modified by Hamilton. In following Virchow's method the organ is so much cut up that in case we are anxious to trace out diseased tracts it is nearly impossible to do so, while by the

method of Peters this can be done perfectly, though the brain as a whole cannot be quite so readily examined as by the former method. Both will be given as concisely as possible.

## CL XXVII.

### Hitzig's Method of sectioning the Brain.

This method must only be used where one does not intend to section the brain for the future study of diseased tracts.

(1) With the brain lying base downwards cut right down to the base of the fissure enclosing the corpus callosum and the fornix on the left hand slightly raise the left hemisphere which is usually the first to be examined. With a sharp knife make a shallow incision along the junction of the corpus callosum with the median surface of the hemisphere. This incision opens the roof of the lateral ventricle.

(2.) In order to further lay the ventricle open continue the incision forward, the whole length of the corpus callosum and outward into the frontal lobe, opening up the anterior horn of the lateral ventricle, while the posterior horn may be opened by continuing the incision out into the tissue of the occipital lobe. Care must be taken not to injure any of the structures that form the floor or walls of the ventricle.

(3.) Lay the left hemisphere on its outer side, and with a long brain knife cut vertically down and through the floor of the ventricle, continuing the incision till the knife reaches almost to the outer surface of the under portion. Be careful not to injure the basal ganglia. The hemisphere can now be turned far outwards on the left side. Open the right ventricle in the same way.

Both lateral ventricles can now be thoroughly studied, their size and contents noted and the choroid plexus investigated. The middle horn of the ventricle can be opened if desired.

(4) Pass the knife through the foramen of Monro and divide

the corpus callosum and the fornix by cutting forward and afterwards. Turn the parts lying posterior to this incision backward, exposing in this way the velum interpositum, which with its choroid plexus can be easily examined.

(5.) Raise this structures and cut the large veins that enter it from the basal ganglia. The velum may then be removed from the pineal body and the corpora quadrigemina.

(6.) This proceeding expose the third ventricle which with the structures that form its walls can be inspected.

(7.) Next divide the posterior pillar of the fornix and turn the corpus callosum, fornix and velum interpositum over to the left.

(8.) Place the left hand under the pons and cerebellum and slightly elevate the vermes. Make a careful incision exactly on the median line through the centre of the vermes. The lateral halves of the cerebellum fall apart and disclose the cavity of the fourth ventricle. Prolong the incision in both directions opening the whole ventricle and the aqueduct of Sylvius. This completes the opening of all the cavities of the brain, and their contents and condition should be carefully noted.

(9.) To further inspect the cerebral hemispheres, support each in turn in the left hand, and with a clean long brain knife divide and subdivide each from before backward, until in the opinion of the operator it has been sufficiently dissected. Carry each incision almost through to the cortex of the under surface.

(10.) In order next to study the basal ganglia it is directed to place the fingers of the left hand under the central portion of the brain and make a number of transverse incisions about 5 mm. apart, beginning at the anterior edges of the corpora striata and passing backward through all the basal ganglia. Each incision is to be carried to a corres-

ponding depth, ending just within the cortex of the under portion. In this way the various structures are held together in their normal position, and still nearly every portion can be examined, and the various cut surfaces compared. The hands can also be folded so as to again restore the brain to its normal shape.

(11.) In order to examine the hemispheres of the cerebellum which have already been separated by the incision that opened the fourth ventricle, it is only necessary to divide each hemisphere by a cut which runs from the 4th ventricle along the middle branch of the Arbor Vitae, extending the same almost to the cortex of its convex surface. The resulting halves can be further subdivided if desired.

(12) Place the pons, medulla and what portion of the cord is attached to the brain, over the index finger of the left hand and divide the various structures by parallel, transverse incisions in the same way that the basal ganglia were divided. The two peduncles of the cerebrum can also be sectioned in the same way.

## CLXXXIII.

Petres Method. The Method of transverse Section.

(1.) Remove the medulla, pons and cerebellum by cutting through the cerebral crura at about their middle point. The portions so removed can be sectioned and examined in the way already given, and the fourth ventricle can be opened by section through the vermes

(2.) Place the brain upon the table vertex downward keeping it in one position while the following transverse incisions are made:

Section I. Through the anterior half of the 3rd frontal convolution

" II. Through the tip of the tempero-sphenoidal lobe and the operculum.

" III. Through the brain immediately in front of the optic chiasm.

Section IV. Through the infundibulum.
" V. Through the corpus Albicans.
" VI. Through the anterior margin of the pons.
" VII. Passes across the front of the angular gyrus.

By the use of this method one is enabled to trace the presence of degenerated tracts from one portion of the brain to another or to trace into the brain degenerated tracts from the cord or medulla. In both the methods given one can very accurately locate the position of lesions and describe the same with exactitude.

## CLXXXIV.

### The Cord.

Section of the cord is made in the same manner as section of the pons and medulla, the incisions being made a few millimeters apart, perpendicular to the long axis of the cord, and not carried quite through its entire thickness.

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# Chapter XIV.

## How to treat wounds received while dissecting or in the course of a post-mortem examination.

## CLXXXV.

Wounds received while dissecting, or while performing an autopsy often lead to serious results unless treated promptly and in the proper way. Such wounds are almost of necessity infected with septic micro-organisms of various kinds which give rise to blood poisoning. No matter how small the wound received it should be immediately disinfected for the smallest pricks with an autopsy needle are likely to lead to as serious results as larger cuts; in fact they often prove to be more serious because free bleeding is not produced.

The following simple directions should be followed. When the wound is a small one, a small cut or puncture it may be cauterized with nitrate of silver stick, or with pure carbolic acid. When it is larger it should be treated in the following manner:-

(1.) Check the venous and lymphatic circulation as soon as possible by applying constrictions.
(2.) Cleanse the surrounding skin with some antiseptic fluid such as bichloride solution, 1 - 1,000, or carbolic acid solution, 1%.
(3.) If necessary to further cleanse the wound thoroughly, enlarge it with a clean sharp knife and allow free bleeding.
(4.) Wash and dress with antiseptic dressings.
(5.) Allow an assistant to finish the autopsy.

Before beginning to dissect, or before every autopsy carefully examine the hands for abrasions, or small wounds. Especially be careful to examine the base and under edges of the finger nails, where not infrequently, small wounds exist that are likely to escape notice. Some operators advise the use of rubber gloves, or finger tips. It is my experience that these do more harm than good because when they become moist with the blood they are very slippery and the danger of becoming wounded by the hand slipping against the knife is greatly increased.

In cases where it is imperative that the operator continue the autopsy after receiving a slight wound, he should, after treating it as first described apply some impervious dressing, or put on a rubber finger tip and proceed very cautiously. One should never close an infected wound with collodion, and even after thorough cleansing, such an application should be avoided for fear of enclosing septic material. Further than this, collodion applied to the skin soon begins to peel off and therefore offers very little protection.

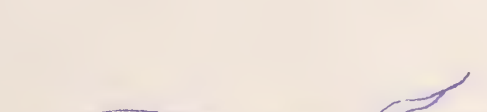
# CLXXXVI.

## Table of Weights and Measurements.

The following figures are taken, with few exceptions from "Daten und Tabellen" by Vierordt, published in Jena in 1893.

| | | | |
|---|---|---|---|
| Brain | Male 1358 grams<br>Female 1235 " | | 15-17-14 × 12.5 cm. |
| Heart | Male 300 "<br>Female 250 " | Length, 8.9 cm. | Width 8.5-10 cm. |
| " | Circumference at base of the ventricles | | 25.8 cm. |
| " | Thickness of left ventricle wall | | 1-1.4 " |
| " | " " right " " | | 0.3-0.5 " |
| " | Depth of left ventricle | | 9.5 " |
| " | " " right " | | 10.0 " |
| " | Circumference of mitral valve | | 10.4-10.9 " |
| " | " " tricuspid " | | 12.0-12.7 " |
| " | " " aortic " | | 7.7-8.0 " |
| " | " " pulmonary " | | 8.9-9.2 " |
| Kidneys | Weight combined, 299 grams. | | |
| | Measure, (11-12) × (5-6) × (3-4.5) cm. | | |
| | Cortex in thickness, 0.4-0.6 " | | |
| | Relation of cortex to medulla, 1 to 3. | | |
| Lungs | Right 513 grams or Male 24 oz. | | Female 17 oz. |
| | Left 441 " | " 21 " | " 15 " |
| | Total 954 " | " 45 " | " 32 " |
| Liver | 1510 grams | | |
| | Width of right lobe, | | 18-20 cm. |
| | " " left " | | 8-10 " |
| | Length of right lobe | | 20-22 " |
| | " " left " | | 15-16 " |
| | Greatest thickness | | 6-9.5 " |
| Ovaries | 7.0 grams | | 4.5 × 2.7 × 1.3 " |
| Pancreas | 66.0-102 " | | 19 × 2.2 × 4.0 " |
| Spleen | 171 " | | 12 × 7.5 × 3.9 " |

| | | | |
|---|---|---|---|
| Suprarenals, | 4.8 - 7.3 | grams, | 4.5 × 2.1 × 0.5 cm. |
| Spinal cord | 33.38 | " | 44.8 cm. Male |
| | | | 41.6 " Female |
| Stomach, | 170.232 | " | |
| Testes | 15-24.5 | " | |
| Thyroid | 30-60 | " | 6 × 3.5 × 2.0 cm |
| Uterus | 110 | " | 9 × 6 × 3.5 in those who have borne children |



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